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MUCOSAL FOLDS AT THE CARDIA AS A COMPONENT OF THE GASTRO-ŒSOPHAGEAL CLOSING MECHANISM

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THE closing mechanism between stomach and œsophagus has been a subject of considerable dispute for more than two hundred years. Whereas most people at the present time agree that competence is produced by the synergistic action of multiple factors, opinion is divided on the exact role of each component. A full understanding of its complex physiological activity has been further complicated by a prodigious number of conflicting theories, some of which are based more on fantasy than fact.

As early as 1815 Magendie suggested that a mucosal fold occludes the cardia. This view, however, has been completely overshadowed by the concept of "the pinchcock action of the diaphragm" (Jackson, 1922); the internal sphincter (Lerche, 1950); the valve of Gubaroff (von Gubaroff, 1886); the cardiac angle of His (His, 1903), and others. Except for indefinite references to vague "folds around the cardia", I could find no conclusive evidence from the literature as to whether these folds existed, and if so, of their exact form and function. As the current theories on gastro-œsophageal competence are unacceptable, I wondered whether these folds might not be an essential feature in the mechanism. With this in view, a series of investigations was carried out in animals and man, to study the structure and function of the mucosal folds at the cardia, if, in fact, they exist at all.

A B

FIG. 665.—A, The cardiac orifice appears as a linear slit with tightly apposed mucosal lips (elderly female patient). B, The watertight seal is open. A definite ridge is present on either side.

ANATOMICAL STUDY

Material.—Specimens were examined from 30 adults (18–80 years) and 115 infants and children (32 weeks prematurity–8 years) of both sexes who died of causes unassociated with the cardiac region. The diaphragm, lower œsophagus, stomach, aorta, and surrounding tissue were carefully removed *en bloc* at autopsy and fixed in 10 per cent formol-saline. A detailed macroscopical dissection and a histological examination were subsequently carried out. Apart

from these specimens, numerous others were also examined consisting only of the lower œsophagus and upper stomach.

Findings.—No conspicuous mucosal folds were found in the first ten dissections and gradually the idea of occluding folds at the cardia was discarded as a myth. However, in the eleventh preparation a perfect watertight mucosal seal was found at the

FIG. 666.—The large voluminous fold projected upwards into the œsophagus from the greater curve side (adult male patient). C indicates the level of the cardia and M the level of the squamo-glandular junction. Opened along the lesser curve.

cardia (*Fig.* 665 A). A 14-mm.-long slightly oblique linear slit extending from behind forwards and to the left represented the cardiac orifice. This was 'opened' carefully, and two definite, well-defined mucosal lips that lay in close apposition could be clearly differentiated (*Fig.* 665 B). These were of about the same size, and in a way resembled the mitral valve. Each fold was round and almost rolled or pad-like at the edge. They projected firmly into the œsophageal lumen as if there had been active tone present during life that maintained the position. Immediately above these folds a slight widening occurred—the potential space of the vestibule.

Another specimen presented an entirely different arrangement. A large voluminous triangular flap of mucosa, based on the greater curve side of the cardia, extended upwards into the vestibule (*Fig.* 666). Although situated above the cardia, this fold consisted entirely of glandular epithelium (confirmed by histology). The squamo-glandular junction was immediately above. From below, this fold plugged the lower œsophagus so admirably that reflux must have been out of the question. On the other hand, it would have caused no real obstruction to œsophageal peristalsis from above.

In the great majority of adult specimens no extraordinary folds were seen, although very marked variation occurred. This was much more so than in the infants. The mucosal fold pattern was quite unrelated to the squamo-glandular junction which is extremely variable. Usually the longitudinal œsophageal folds widen in the vestibule and terminate at or just distal to the cardia in small triangular mucosal pads. As the junction of squamous and glandular epithelium is above the cardia in most people, these elevated pads usually consist of glandular epithelium. Other gastric folds arise from about 3–15 mm. distally as broad raised ridges. Uncommonly, the œsophageal folds continued with the gastric folds, but even if this happened, some localized thickening was present at or immediately distal to the cardia. Sometimes the œsophageal folds narrowed and disappeared at the cardia, whereas the gastric folds began as small elevations. In some specimens the folds were unimpressive, or almost non-existent. The fact that no uniform pattern, size, or form of folds at the cardia was found was considered to be in keeping with the difference in functional behaviour as evidenced by clinical and special examinations.

The observations in this small series strongly suggested that, in at least some people, a definite arrangement of special mucosal folds exists. These people either possessed exceptional qualities at the gastro-œsophageal junction which others lacked, or else this is a normal phenomenon that is absent in the majority at the time of post-mortem examination, but preserved in the few. The latter was considered to be more likely. These folds might have been disturbed at the time of removal of the specimen from the body at autopsy; or they might have disappeared as a result of post-mortem changes in the functional anatomy, which could account for the fact that many workers in the past either denied the existence of these folds or speculated on their form and position. The first possibility was easily and quickly excluded by very careful dissections at autopsy with the organs undisturbed and in situ. Even under these conditions the gastro-œsophageal mucosa was smooth, glazed, and flabby; no significant folds were found in any of the subjects thus examined.

On the assumption that special folds are present during life that rapidly disappear after death, the gastro-œsophageal junction was examined in a series of patients at operation.

MUCOSAL FOLDS IN THE HUMAN AT OPERATION

Method.—Twelve patients, including 2 females, who were subjected to partial gastrectomy for benign ulceration of the lower stomach or duodenum were studied (Botha). The ages varied from 22 to 58 years. The general anæsthetic in every patient included relaxants; the position was supine. The procedure followed that of a routine gastrectomy and the stomach was mobilized up to the stage of anastomosis. This organ was then adequately packed off with mops to avoid any possible soiling of the surrounding tissues. The finger was inserted

through a small hole in the anterior gastric wall distal to the anticipated suture line. The cardiac orifice, folds, fundus, crura, and lower œsophagus were very carefully and gently palpated taking 5 to 10 minutes over the examination. At first it was very difficult really to feel the anatomy, but after the first three attempts I was satisfied that the finger could record an almost ' visual ' impression of the cardiac orifice and associated structures. This ' visual ' impression was schematically drawn on paper together with any relevant data immediately after completion of the operation. The following observations were made.

Findings.—There was evidence of occluding mucosal folds at the cardia in every patient, although hardly any two of them presented the same pattern.

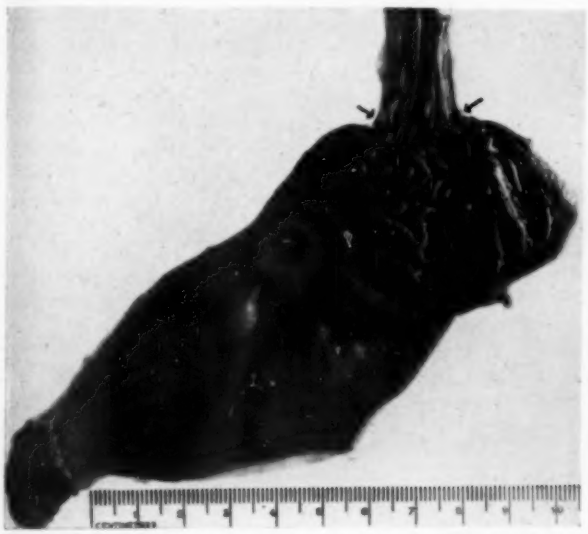


FIG. 667.—The striking cardiac folds in the stomach of the tortoise. The arrow points to the squamo-glandular junction.

In 2 patients the apposition was so perfect that the cardiac orifice was located only after some search: in one it was found only after the anæsthetist had passed down a Ryle's tube. It should be appreciated that the location of the cardia was conducted with the utmost gentleness since force would have vitiated the very object of the investigation.

In the majority of cases the orifice was easily located and existed as a dimple on the lesser gastric curve. It always surprised me how low down the cardia is. One is inclined to search for it at a much higher level. Whereas it was difficult to locate the orifice if the finger advanced in the line of the gastric curve, it was relatively easy to find if the finger was directed at a right angle to the lesser curvature. The lower œsophagus immediately above the cardia felt ' closed ' to the approaching finger-tip. This positive ' gripping ' tone was slight, but undeniably present, and unrelated to extrinsic structures. In a few cases it appeared to be wide open, although voluminous folds of mucosa were easily palpable. The cardia on these occasions was flabby, patulous, and apparently devoid of tone. On the greater curve there was often a baggy crescentic fold that hung on the orifice like a curtain. In most of the patients one had the impression that some loose radiating folds converged on the orifice or that a few concentric floppy mucosal ridges guarded the opening from below. A constant feature on the lesser curve side of the cardia was a cushion-like resistance that could easily be detected by the moving finger over an area of 5–10 mm. Externally the stomach revealed no groove or constriction at this site. In one patient the mucosal orifice was small and tight, and although it only admitted one finger, it was soft, freely mobile, and could be stretched. The mucosal edges on palpation were identical with those in *Fig.* 665.

It cannot be over-emphasized that the utmost care and gentleness should be exercised in the palpation. A quick casual examination would only leave doubt as to the existence of these folds, and

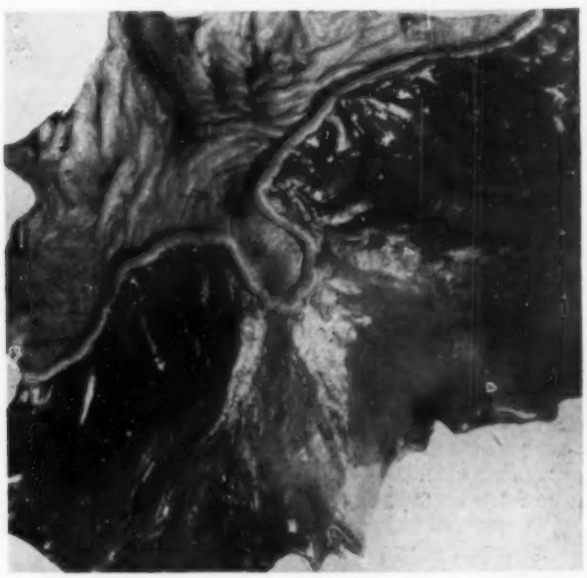


FIG. 668.—A conspicuous fold separates the squamous from the glandular epithelium on the interior of the rat's stomach. The arrow points to the cardiac orifice.

their form and exact localization cannot be assessed by rough palpation. Much more important, however, is the fact that the ' tone ' of these folds appeared to change after some manipulation. Sometimes the cushion-like pads lost all resistance, the folds smoothed out, and the orifice became wide open and lax—a totally different picture from what it had been. The cardia was always more patulous and the folds were less pronounced when a Ryle's tube had been passed pre-operatively. The tube, when present, was pulled up before the beginning of each investigation.

These findings strongly supported the existence of physiological mucosal folds at the cardia. It remained to be seen to what extent these conclusions could be supported by data derived from the examination of other mammalian stomachs.

COMPARATIVE ANATOMY

The mucosal-fold pattern at the gastro-œsophageal junction was studied in 21 different species: dog-fish, frog, tortoise, fowl, ferret, dog, fox, pig, sheep, ox, horse, monkey, mouse, rat, hamster, bat, guinea-pig, rabbit, mole, hedgehog, and cat (Botha). These folds were squamous or glandular. The type of epithelium bore no relation to the level of mucosal change.

The most convincing example of occluding cardiac folds was found in the tortoise (*Fig.* 667). The voluminous folds of gastric mucosa arising almost abruptly at the squamo-glandular junction are thick,

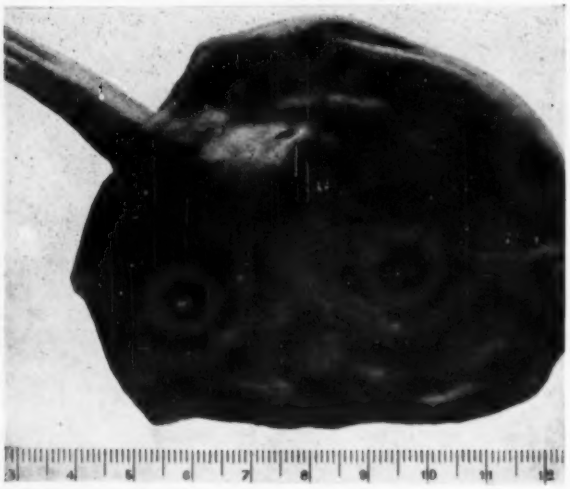


FIG. 669.—The smooth, glazed appearance of the mucosa in the fox corresponds closely to that which is commonly seen in the human stomach at autopsy.

fleshy, and cushion-like. These wavy longitudinal folds gradually fade away into a relatively smooth gastric mucous membrane. Near the cardia they are especially bunched up and tortuous. In the absence of a diaphragm and without macroscopical signs of an internal sphincter in the lower œsophagus, these folds constitute the major factor in the tortoise which prevents the forcible ejection of food when the stomach contracts.

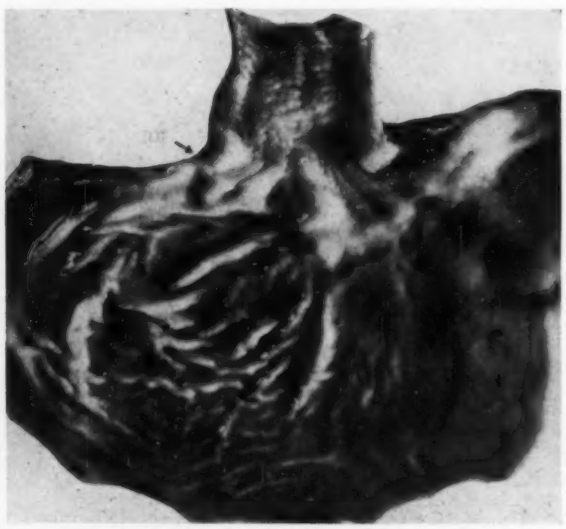


FIG. 671.—A slightly puffed-up mucosa is present at the cardia in the cat. The arrow indicates the anatomical cardia.

Such folds are completely absent in the guinea-pig and mole. The mucosal change is so abrupt 1 mm. above the cardia that a fringe of stratified squamous epithelium projects through the cardiac orifice into the stomach. The glandular epithelium below this is perfectly smooth. The gastric mucosa in the bat forms an appreciable conical papilla at the cardiac orifice. The apposition of the edges is so watertight that this 'cork' effectively blocks the gastro-œsophageal junction when the animal is hanging in

FIG. 670.—Small rugose folds are present at the cardia in the monkey. The lower œsophagus widens and the squamo-glandular junction (arrowed) lies immediately above the folds which consist of glandular epithelium.

FIG. 672.—The pig presents a lax flap of squamous epithelium at the cardia.

the peculiar upside-down position which is characteristic of the species. The squamous epithelium in the mouse, rat, and hamster extends well down into the stomach and joins the glandular epithelium at the distal margin of a pronounced oblique ridge that divides the stomach roughly into an upper fundic

and lower pyloric region (*Fig.* 668). The physiological importance of this ridge or fold is not certain. No sign of any folds was found in the ferret and hedgehog.

The cat, dog, and monkey presented almost identical features. The mucosal change in every specimen took place immediately above the cardia and was very constant. Some vague irregular and inconsistent folds were present at the cardia in some animals, whereas in others, such as the fox, a smooth, glazed mucosa reminded one of the common appearances in the human stomach at autopsy (*Fig.* 669). At the cardia in the monkey and the cat the glandular epithelium had a puffed, thickened, slightly rugose appearance (*Figs.* 670, 671), but no special folds or redundancy of mucosa were ever seen here. The cardiac orifice in the pig was reduced to a slit with a curtain-like fold projecting down from the greater curve side (*Fig.* 672). In one specimen this fold was 15 mm. wide. Numerous irregular criss-cross folds are present in the horse and form an almost honeycomb-like effect at the cardia, completely obliterating the lumen. The gastro-œsophageal junction in ruminants is, with the exception of the lips of the œsophageal groove which characterize specialized function, singularly free of mucosal folds.

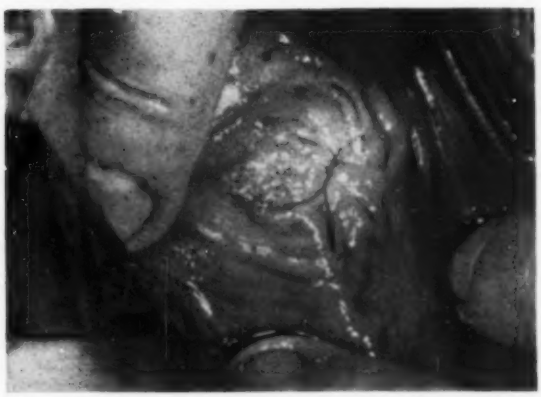
The rabbit's stomach, examined several hours after death, presented a very smooth atrophic looking mucosa around the cardia with no suggestion of any folds. When the stomach was examined immediately after death, however, the difference was remarkable. A circular serrated fold of glandular epithelium prolapsed down into the stomach to form a perfect mucosal rosette at the cardia. The orifice was tightly closed, the conical mucosal papilla was air-tight; the occlusion was at or below the cardia (*Figs.* 680 C, 686). This striking contrast in the rabbit strongly suggested that the appearance of functional anatomy and post-mortem or cadaver anatomy might be totally different. The next step was to confirm this by a series of physiological experiments in animals.

COMPARATIVE PHYSIOLOGY

The form and activity of the mucosal folds at the cardia were studied in 13 different species. In order to interfere as little as possible with œsophageal and cardiac tone and motility, operations were performed at the lightest plane of anæsthesia (Botha and Nunn, 1957). Intravenous or intraperitoneal pentobarbitone or avertin, supplemented by nitrous oxide and oxygen where indicated, were satisfactory. No relaxants, atropine, or opiates were used. At laparotomy the stomach was widely incised, and the cardia exposed. At other operations, thoraco-laparotomy exposed the stomach, cardia, and lower œsophagus. Swallowing, primary, secondary, and spasmodic peristalsis were initiated by squirting water into the pharynx and by distending œsophageal balloons. Sometimes the movements at the cardia were so rapid and the folds changed so quickly that it was impossible for the human eye to comprehend fully what was happening. This dynamic activity was therefore recorded on normal speed, as well as on slow-motion cinematography films in 10 different species, and was studied afterwards. Although these films are a credit to the technical ability of the photographer the 16-mm. frames do not enlarge well enough for all the details to be seen in the pictures reproduced in this publication. There was, however, no other way of securing some of these photographs. Although I apologize for their quality, I hope they will stimulate interest, especially amongst physiologists.

A

B



C

FIG. 673.—A, The cardiac orifice in the dog during life. Note the full rosette. (Ciné-film.) B, The stomach wall stretched. The mucosa becomes smooth except at the cardia where the rosette is still well marked. (Ciné-film.) C, The cardia is tightly closed and the mucosal rosette, which can still be recognized, projects into the lumen as a conical papilla. Competence is still perfect and the seal watertight, despite complete division of the diaphragm and obliteration of the angle. Compare these cinematograph pictures with *Fig.* 669.

These folds are now briefly described; full physiological discussions are now in publication.

The striking difference in the functional anatomy and the macroscopic appearance of the dissecting room specimen was the outstanding feature of this investigation. Not only did the specimen look entirely different, but it also acted in a way that at times was completely unexpected.

In the empty stomach of the dog, the animal which has been most extensively used for experimentation

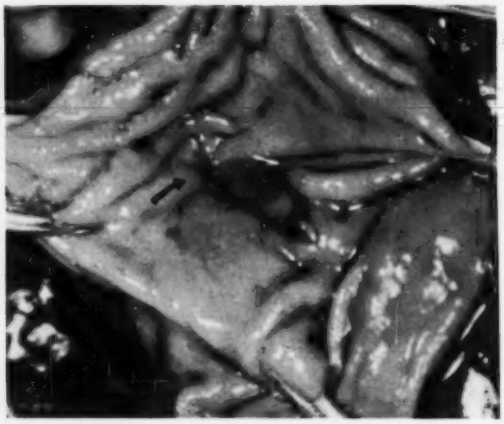


FIG. 674.—The mucosal rosette in the cat is less marked than in the dog. No squamous epithelium can be seen. (Ciné-film.) Compare with *Fig.* 671.

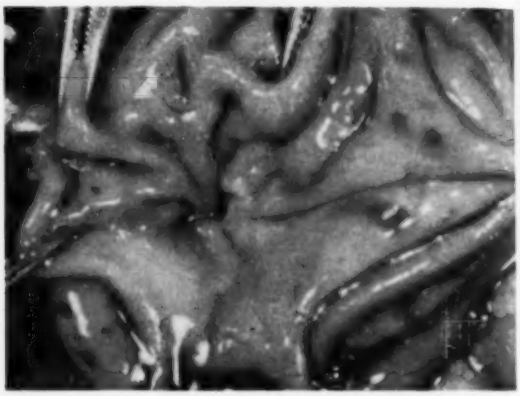


FIG. 675.—The sharp edge of the tonic cardiac fold in the monkey is quite obvious on the left side where the stomach wall is put on the stretch. (Ciné-film.) Compare with *Fig.* 670.

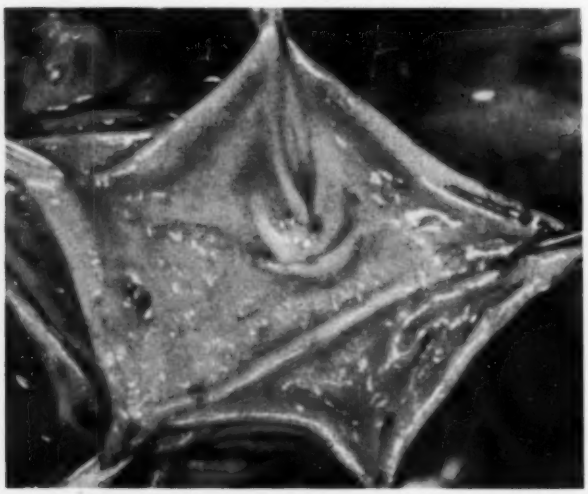
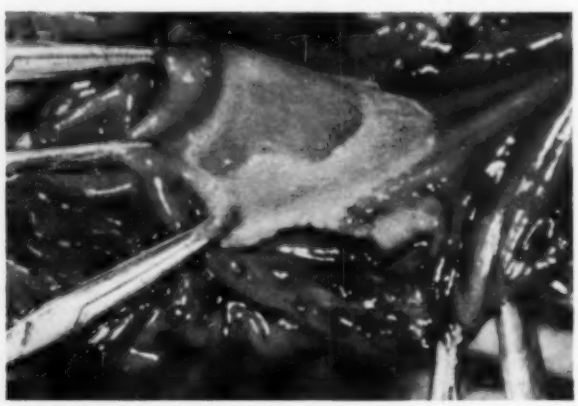


FIG. 676.—The ferret sometimes presents a double rosette. In this picture the second fold has almost been drawn out. (Ciné-film.)

FIG. 677.—The guinea-pig cardia is normally closed flush with the gastric surface. On vagal stimulation it opens and presents a 'golf-hole' opening, indicated by the arrow. (Ciné-film.)

A

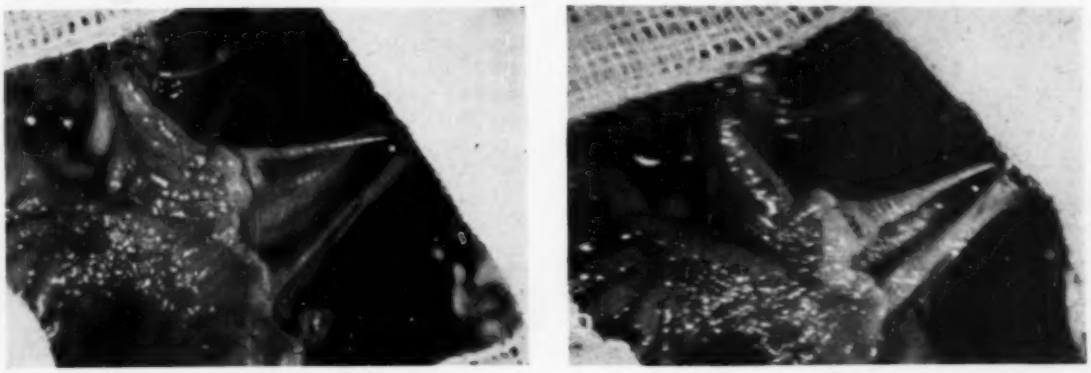


B

FIG. 678.—A, The cardiac orifice presents in the pig as a watertight linear slit that is hardly visible. B, The orifice opens on vagal stimulation. (Ciné-films.) Compare these figures with *Fig.* 672.

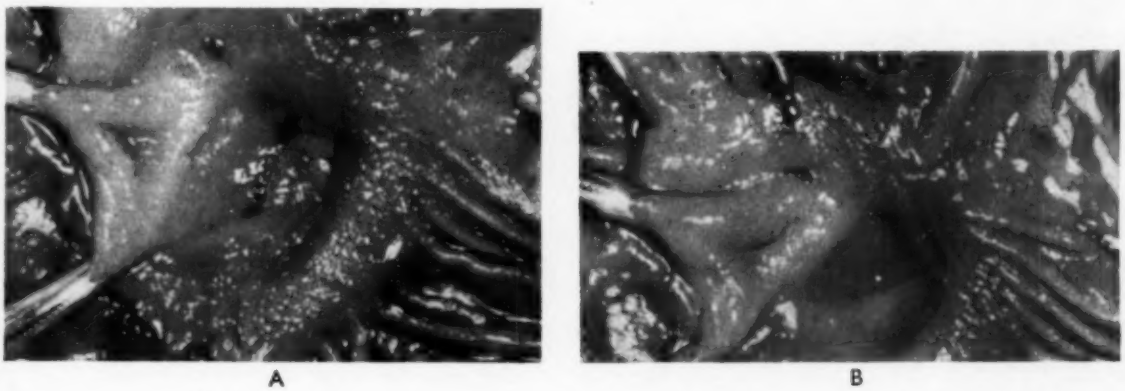
on the cardia, it was sometimes impossible to locate the cardiac orifice. The irregular folds were lying close together in a criss-cross fashion and constantly changed in shape, form, and position (*Fig.* 673 A). These variations appeared to be mainly dependent on the alterations in tone of the surrounding musculature. However, independent tone was present in the folds, which brought about a certain amount of movement, irrespective of external factors. If the stomach wall was put on the stretch away from the cardia, the folds gradually smoothed out until ultimately only a rosette of folds was present around the cardiac orifice, radiating outwards to a smooth periphery (*Fig.* 673 B). If the wall was stretched further the rosette tended to disappear, except immediately around the œsophageal opening. Slight elevated folds bulged into the stomach and formed a large convex cone with the tightly closed cardiac orifice in the centre (*Fig.* 673 C). Only with some difficulty could the cardia be opened. It was always completely closed despite removal of the diaphragm and obliteration of the cardiac angle. One dog was operated on within an hour of a big feed and the stomach was found to be full of food. The peripheral gastric folds were shallower and farther apart, but the cardiac folds represented the same picture as when the stomach was empty.

The folds in the cat were identical with those in the dog, perhaps not so closely packed but of the same pattern (*Fig.* 674). In the monkey (Rhesus) the folds were smaller, wider apart, and the rosette at the cardia was not so well marked. The gastric mucosa formed a definite circular ridge immediately below the squamo-glandular junction, and at the cardia it was bunched up and closed the orifice from below so that it could not be seen unless the gastric wall was pulled peripherally (*Fig.* 675). If the finger was introduced into the lower œsophagus and withdrawn quickly, the cardia would slowly contract to its previous state of closure. The mucosal edge



A B

FIG. 679.—A, The cardia and lower œsophagus of the rabbit slit open. The vestibule and sharp mucosal ridge at the cardia are well defined. B, Bilateral cervical vagal stimulation. The massive sphincter, bunching up the mucosal fold and decreasing the size of the vestibule, is quite striking. (Ciné-films.)



A B C

FIG. 680.—A, The tightly closed cardia in the rabbit. The gastro-œsophageal vestibule telescopes upwards. B, Complete retraction of the cardia during vagal stimulation. C, The 'resting rosette' in this figure is much smaller than the 'prolapse rosette' which appears at the end of the caudal descent of the vestibule after swallowing or vagal stimulation. (Ciné-films.)

imparted a feeling of positive resistance. In not one instance was there a passive arrangement of these folds; they were always controlled and activated by some inherent tone. In all three species mentioned it was noticed that repeated palpation and manipulation of the folds around the gastro-œsophageal junction resulted in a patulous orifice, which sometimes remained open for a while, apparently without tone. This was always temporary. Within half a minute or so, tone would return, the

FIG. 681.—The stellate appearance which is commonly seen at the site of the cardiac mucosal folds. (Adult patient.)

folds would gradually assume the old position, and 'the gate was closed' as before. Single mucosal folds did not exist. There was never the slightest suggestion of a mechanical flap or a valve operating at the gastro-œsophageal junction and blocking the orifice by chance.

The ferret and hedgehog presented roughly the same mucosal patterns. The folds were finer and extremely abundant. The rosettes were often formed by a double row of radiating folds which closed the cardia effectively (*Fig.* 676). The guinea-pig cardia was closed by a thickened circular elevation of glandular mucosa that presented immediately distal to the squamo-glandular junction. There were no rugose folds present. The sharp edge of the mucosa, where it was in apposition at the cardiac orifice, could be seen clearly when the stomach wall was pulled away from the cardia (*Fig.* 677). The cardiac orifice in the pig presented as an elongated slit of varying length. No mucosal rosette was seen in any of the experimental animals. Instead, the right and left lesser curve walls approximated very closely (*Fig.* 678 A). A curtain-like fold attached to the greater curve actively closed the cardiac orifice in one animal. This fold was in a state of constant varying tone and changed slightly in position, shape, and approximation to the opposite side (*Fig.* 678 B).

The mucosal pattern in the small rodents was of an entirely different nature. The peculiar ridges formed where the squamous and glandular epithelium joined were very prominent around the cardia. On the dorsal and ventral wall these appeared to form elevated pads that normally protruded inwards towards the cardiac orifice. The folds sometimes bent towards the œsophageal opening and occluded it completely from one's view. On bilateral vagal stimulation, the folds drew nearer to each other and pressed tightly into the cardiac orifice. During the passage of a peristaltic wave they separated, allowed the bolus to pass, and then reverted back to the previous position.

The rabbit presented the most perfect rosette that was found in any of the species. (The physiological activity of the cardia in this animal is remarkable, if not unique, and must be seen to be fully appreciated.) As a result of the constant activity, particularly during acts of swallowing or vagal stimulation, the mucosal pattern changed from moment to moment in a most fascinating fashion (*Fig.* 679). A serrated circular elevation of mucosa surrounded the cardiac orifice to form an almost flower-like rosette that projected slightly into the stomach. The cardiac orifice was tightly closed, and this watertight closure was distal to the true cardia. The greatest change in the mucosa took place during the rhythmical telescoping of the cardia. As it went upwards the rosette followed, became obliterated, and the orifice was closed but smooth (*Fig.* 680 A). On vagal stimulation, the œsophagus contracted and the cardia completely disappeared as a black hole in the stomach wall (*Fig.* 680 B). The following moment the lower œsophagus projected down, the cardia prolapsed into the stomach, the rosette appeared, and finally the mucosa completely everted with a forceful thrust at the end of the descent. It immediately inverted again and the cardia retracted, leaving the serrated rosette in its resting position (*Fig.* 680 C). Minor variations occurred according to the age and kind of rabbit, but the basic structure and behaviour of the mucosal pattern were the same in more than 40 consecutive experimental animals.

These animal experiments left no doubt that physiological folds at the cardia not only exist, but that they fulfil an essential function in the gastro-œsophageal closing mechanism. It further proved beyond doubt that the anatomical appearances of lifeless specimens in animals and man bear no resemblance to what these same tissues and organs look like under physiological conditions. Further proof of the physiological presence and watertight closure of the cardiac folds in man is satisfactorily demonstrated by radiological examinations of this region.

RADIOLOGICAL OBSERVATIONS

At barium swallow one usually sees the abrupt termination of the longitudinal œsophageal folds at the cardia. Sometimes, immediately after a bolus of barium has passed into the stomach, an irregular star-shaped or double crescentic shadow can be identified at the cardia. This is due to a lingering barium layer caught by the mucous folds at the cardiac orifice after passage of the bolus (*Fig.* 681). This is *not*, as is commonly thought, the site of mucosal change, to which it bears no constant relation. If, at dissection, the naked eye can just differentiate the change in mucosa, it is difficult to

FIG. 682.—A slight constriction occurs at the site of the mucosal folds. Note the long abdominal œsophagus (from the level of the clip, which was attached to the undersurface of the diaphragm (Botha, 1957), to the arrow). (Adult male.)

FIG. 683.—The characteristic 'empty segment'. The clip is fixed to the abdominal surface of the hiatus immediately in front of the œsophagus. (Adult female.)

understand how radiographs can localize this level with such certainty.

These folds are best observed with the patient erect and in the left oblique position. The stomach should preferably be empty. The œsophageal folds are traced down to cardiac level where they suddenly radiate or end. Sometimes a very localized narrowing occurs at this level, clearly dividing the œsophagus and the stomach (*Fig.* 682). These radiological patterns change from moment to moment in the same patient. In some patients they show up much better than in others; in some they may be entirely absent. They may be quite definite on one occasion and absent later in the same patient. Although they are mostly star-shaped, crescents, horseshoes, and other forms have also been seen. Exactly the same appearances were observed in children.

The most outstanding radiological feature at the lower end of the œsophagus has always been the 'empty segment' above the cardia (*Fig.* 683). I have never yet, in any *normal* patient, seen this 'empty segment' fill from the stomach side (excluding the momentary small refluxes which are obviously due to a temporary relaxation in tone). Despite pressure on the abdomen, a steep Trendelenburg position, or forced inspiration, the empty segment stays empty and the barium-filled stomach shows an uninterrupted smooth outline. No diaphragm, sphincter, or anything else could on its own produce this smooth, watertight seal except mucosal apposition; and we have already seen that this apposition is not the result of chance, mechanical mechanisms, or angulation.

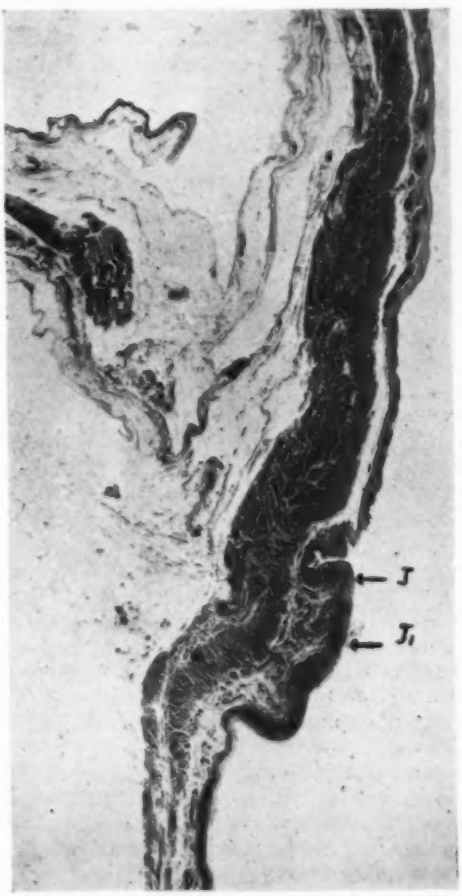


FIG. 684.—A longitudinal section through the hiatus and gastro-œsophageal junction in an adult female. Note the thickened muscle layers in the lower œsophagus and the redundant mucosal fold at the cardia. The arrows J–J_1 indicate the epithelial change.

HISTOLOGY

All the above investigations suggested that the physiological mucosal folds at the cardia in both man and animals are in a state of positive tone and

constant activity which are mainly influenced by the surrounding musculature. However, the experimental observations also favoured a definite intrinsic force in the folds themselves, quite independent of the stomach wall. The muscularis mucosæ is the only structure that may be responsible for this. To ascertain its importance, histological sections were

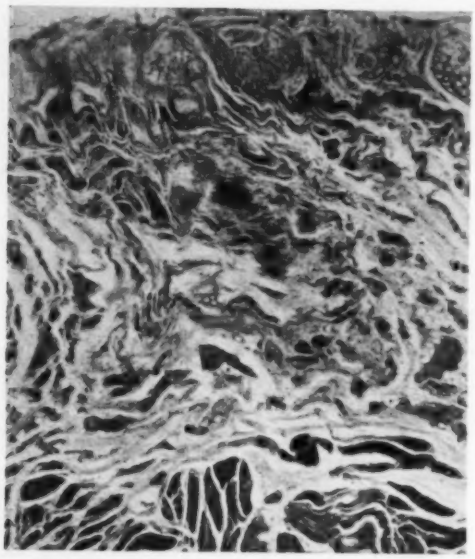


FIG. 685.—Enlargement of a section in *Fig.* 684 (J–J_1) to show the well-developed muscularis mucosa.

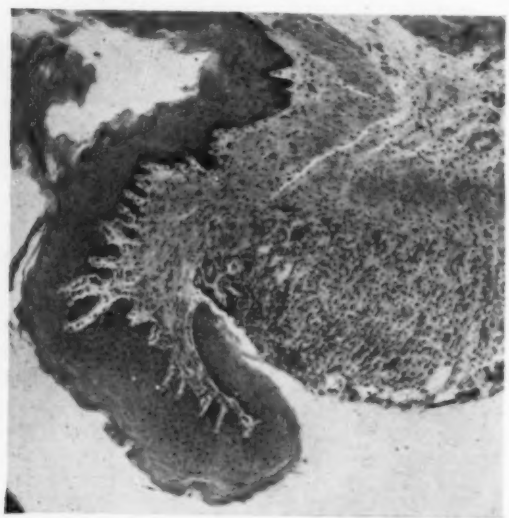


FIG. 687.—The well-developed muscularis mucosa is mainly attached to the base of the mucosal fold in the rat.

studied from the gastro-œsophageal region in the human patient as well as in animals.

The muscularis mucosæ was definitely thickened at the cardia. This varied widely. In some patients no obvious thickening could be seen, whereas in others it was two or three times as thick at this level (*Fig.* 684). The smooth muscle-fibres themselves showed no change, but the whole layer was more irregular at this site. Numerous fibres were attached to the epithelial lining as well as to the submucous and muscle layers, which resulted in an almost tethered appearance. Sometimes the muscularis widened and divided into numerous bundles which formed an irregular whorl, or splayed out (*Fig.* 685) towards the epithelial covering. Some animals also

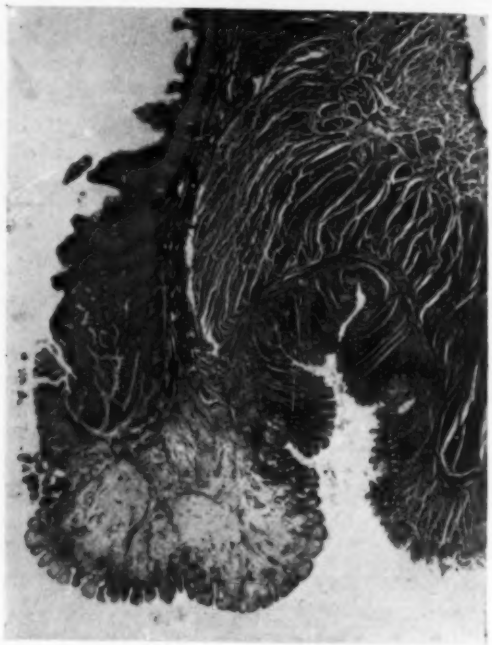


FIG. 686.—The cardiac mucosal fold in the rabbit is almost entirely controlled by the powerful muscularis mucosa.

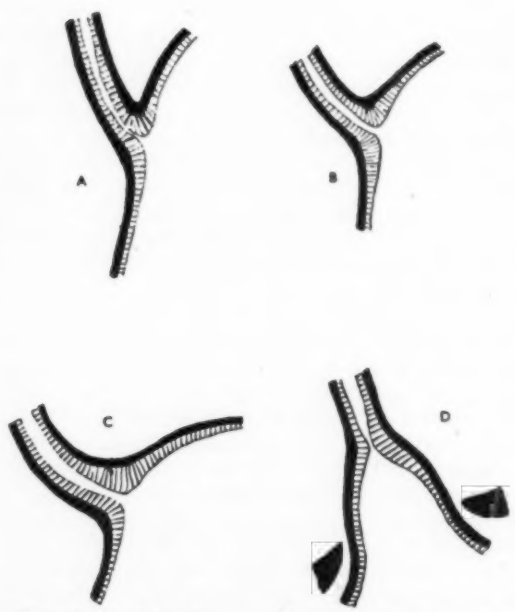


FIG. 688.—A, B, C, The normal human cardiac angle varies from very acute to very obtuse. As the intragastric pressure is evenly distributed, the 'opening force' at the cardiac orifice is small. D, In hiatus hernia the opposite holds; a small force tends to open the orifice.

showed a thick muscularis near the cardia. In the rabbit (where the mucosal rosette is subjected to excessive movement) the muscularis was particularly well developed and splayed out in radial strands to be attached to the fold (*Fig.* 686). This was equally

well demonstrated in the rat (*Fig.* 687). These appearances very strongly suggest that the muscularis mucosæ is specially adapted to control and maintain independent tone in the folds that guard the cardiac orifice.

DISCUSSION

Even in the lowest vertebrate class where the upper gastro-intestinal tract consists of a straight tube, evidence suggests that a valvular mechanism exists at the lower end of the gullet. The conspicuous fringed papillæ in the œsophagus of the dog-fish probably facilitate the backward flow of ingesta. In the great basking shark (Selache) the homologous processes near the cardia acquire unusual length, dividing and subdividing as they extend into the lumen so that the cardiac opening is surrounded by ramifying tufts directed towards the stomach. This valvular mechanism is intended to prevent the return of such fish or molluscs as may have been swallowed alive and are uninjured by the small pointed teeth of this great shark. It is probably one of the most simple known mechanisms of ' cardiac control ' and constitutes the anatomical and physiological precursor of the occluding mucosal folds at the cardia in higher orders. The variations through the animal kingdom are legion ; many of the final answers are still outstanding.

In this paper the literature has purposely not been referred to. Instead, I have summarized a few observations on the mucosal folds as the problem occurred to me in logical sequence during the course of the investigations. As it is impossible to discuss fully the other intricate aspects of the fascinating closing mechanism between stomach and œsophagus, I have confined myself to the briefest description of the folds only. A more detailed and fuller account of other aspects of the closing mechanism are described elsewhere (Botha, 1957).

The following personal conclusions are the result of my own observations :—

1. There are at the human gastro-œsophageal junction during life occluding mucosal folds of varying size, shape, and position. The majority resemble a rosette.

2. The great variation in the anatomy and physiology of the gastro-œsophageal junction in different species makes it unwise to compare one animal analogously with man. Only by correlating underlying principles in the mammalian series may one arrive at a better understanding of cardiac function.

3. The idea of a mechanical valve or flap is illogical. In not one animal in any of the species was there even the slightest suggestion of this ; neither was there any proof for it in the human.

4. These folds are not passive, but contain an inner contractile element, the muscularis mucosæ, which is specially adapted at this site to maintain a constant active tone.

5. The folds are normally in close apposition at the cardiac orifice and effect a watertight seal from below.

The folds are drawn together and apposition is maintained by the inferior œsophageal sphincter. The evidence in animals and man that a definite physiological sphincter exists has been so overwhelming in all the lines of investigation that one must accept its presence (Botha, 1957). That this sphincteric segment stretches over some distance immediately above the cardia has been conclusively proved by a combined ciné-radiographic and manometric study of the gastro-œsophageal junction (Botha, Astley, and Carré, 1957). The folds alone are too weak to withstand any substantial strain. The internal sphincter by itself, however strong, will always leave a small conical space where the œsophagus joins the stomach, readily detectable radiologically, and a constant invitation to reflux during sudden sharp rises of intra-abdominal pressure. The pads of mucosa fill this potential space and close the cardiac orifice flush, so that the pressure is dissipated evenly along the stomach wall. In this way pressures of 100 and 120 mm. Hg are resisted without the slightest sign of incompetence, yet the mechanism is flexible enough to allow normal peristalsis and the effortless eructation of gas.

These two factors, *the mucosal folds and the internal sphincter, act together in perfectly balanced harmony. Together they form the closing mechanism between the stomach and the œsophagus.*

The diaphragm is not part of the closing mechanism. It is, however, the most important single factor in maintaining the abdominal œsophagus and cardia in their proper positions, and the natural closing mechanism can only act with optimum efficiency if the normal anatomical relationships are preserved. In some people, and especially in children, the œsophageal hiatus is relatively small and the crura well developed. The diaphragm then becomes an important accessory mechanism which assists the internal sphincter in maintaining a closed cardiac orifice, particularly when a greater strain is imposed on it during coughing, deep inspiration, etc. With advancing age, the hiatus widens and the crural muscle becomes attenuated. The phreno-œsophageal membrane is thin and overstretched so that the abdominal œsophagus is less securely fixed. Excessive sliding, once started, rapidly gets worse. The cardia becomes elongated and gradually it is displaced into, and later through, the hiatus into the posterior mediastinum. If the intra-abdominal pressure then rises, a vicious state of affairs will exist. There is no even distribution of pressure ; instead the gastric contents rush into the funnel where the stomach passes through the diaphragm, fill the stomach portion above, and if the force is great enough the deranged mechanism gives way to free reflux. Not only has the closing mechanism been deprived of the important accessory control of the diaphragm, but under these circumstances this structure may on contraction add to the incompetence, firstly by forcing more gastric contents into a tense stomach loculus above, and secondly by causing greater elongation of the cardia as the diaphragm descends.

The cardiac angle is a characteristic feature of the gastric organ which is quite incidental to this problem, and has no part in the normal closing mechanism (Botha). It becomes evident firstly in amphibians, is well marked in reptiles, and varies in mammals according to the functional specialization of the organ. This angle is not formed or maintained by

the oblique fibres. Gastric dilatation is obvious in the human embryo by the end of the third week. By the sixth week the typical shape of the stomach is well established and by the end of the second month the fundic outgrowth appears (Botha). All this takes place long before the oblique fibres are developed. They are therefore the result of the asymmetrical development of the organ, and are not primarily a sphincter. Action as a sphincteric bundle has been highly overrated, yet I do not deny that these fibres may assist in approximating the greater and lesser curve walls of the orifice, thus narrowing the upper portion of the stomach. In the resting stomach these fibres have no part in gastro-œsophageal competence.

The cardiac angle is extremely variable and depends on the age, posture, and build of the patient as well as on the fullness of the organ. It may be very obtuse or very acute, and competence does not depend on the acuteness of the angle, provided that the force which acts on the cardia is evenly distributed over the stomach wall (*Fig.* 638). When, however, the gastro-œsophageal junction is converted into a funnel, as in hiatus hernia, the force acts mainly and directly on the cardiac orifice and reflux is inevitable if the force is strong enough. In the repair of a hiatus hernia the cardia is restored to its normal position below the diaphragm. The improved competence is not due to the ' sharp ' angle or renewed valve, but to the fact that the funnel is reduced and the closing mechanism returned to a position from where it can act with much greater advantage.

The importance attached to the valve effect of the cardiac angle is mainly derived from manometric studies on the cadaver, or even worse, on isolated gastric organs. Nothing but mechanical properties could be displayed by these dead and atonic sacs. The gastro-œsophageal junction is a dynamic living mechanism which is continuously influenced by peristalsis, posture, respiration, cardiac pulsation, and stomach fullness, to name but a few of the many controllable and uncontrollable factors. The following two examples are typical of many which refute this misconception :—

1. In near-total gastrectomies, the angle is completely obliterated, yet cardiac competence is often as perfect as in the normal individual (Botha).

2. In some cases of hiatus hernia, the characteristic ' empty segment ' is clearly demonstrated above the supra-diaphragmatic stomach loculus. Although the valve has disappeared, reasonable competence even in this position is maintained by the synergistic action of the folds and the sphincter.

One fundamental concept must be appreciated : the cardia, in its structure and function, shows wide variation. There is no mystery in this. Gall-bladders, colons, and appendices do not all look the same, neither do they act in the same way. Why should the appearance and response of this part of the body be uniform ? A tight hiatus might be of supreme importance in one person, but of little consequence in another. The sphincter might be so well developed as to be macroscopically evident ; on the other hand, its feeble contraction might be hardly noticeable. Although the underlying principles are the same, the keyword should be variation—not similarity.

SUMMARY

The closing mechanism between stomach and œsophagus has been the subject of controversy for more than two hundred years. As the current theories are unsatisfactory, a series of anatomical, physiological, and radiological investigations was carried out to study this problem.

Dissections of the gastro-œsophageal junction in adults and infants revealed that special mucosal folds were present at the cardiac orifice of some people. As these folds apparently disappeared after death, due to post-mortem changes, the cardia was examined at operation in patients who had a partial gastrectomy, with further evidence of occluding folds. Various types of folds were seen in animals immediately after death, but there was no evidence of these after a few hours. The functional activity of these folds was proved by direct examination of the gastro-œsophageal junction in anæsthetized animals. These folds appeared to have active independent tone which could only have been due to a well-developed muscularis mucosæ, as was proved histologically.

The mucosal folds at the gastro-œsophageal junction are actively drawn together by the internal œsophageal sphincter to produce a watertight seal at the cardia. These two factors, the folds and the sphincter, act together in perfectly balanced harmony to form the closing mechanism between stomach and œsophagus. The diaphragm is an important accessory mechanism and maintains the normal position of the gastro-œsophageal segment so that the closing mechanism can act with the greatest advantage. The angle is an organ characteristic which, as such, plays no part in the closing mechanism. A mechanical valve does not exist.

Acknowledgements.—I wish to thank Professor F. A. R. Stammers for his constant encouragement and advice in preparing this paper ; Mr. B. N. Brooke for his patient co-operation in the theatre ; Mr. T. F. Dee (clinical photographer to the Queen Elizabeth Hospital, Birmingham) for the photographs ; and the Endowment Fund for a generous grant.

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SOLITARY LIVER NODULES (FOCAL CIRRHOSIS)

TWO CASE REPORTS

By J. B. SHERRY, I. B. MACDONALD, and W. ANDERSON

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WHETHER a solitary liver nodule encountered incidentally during laparotomy represents a metastatic carcinoma, a primary liver tumour, or a lesion herein designated 'focal cirrhosis' is a problem occasionally facing the surgeon. The following case reports are presented to draw attention to the salient pathological features of the last-mentioned condition.

CASE REPORTS

Case 1.—Mrs. T. M., aged 29. During the six weeks preceding this patient's operation, she suffered from repeated attacks of gall-stone colic. The last bout of right upper quadrant abdominal pain was associated with mild jaundice, dark urine, and light stools. Slight tenderness was present over her gall-bladder region, but no masses were palpable. A diagnosis of cholecystitis with cholelithiasis was made, and it was thought likely that stones were also present in the common bile-duct. Chemical tests of liver function and urine analyses tended to confirm this clinical diagnosis. There was no history of liver toxin being absorbed.

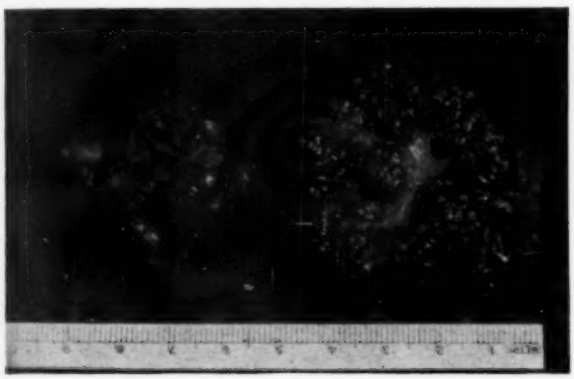

FIG. 689.—Bisected tumour removed from the liver.

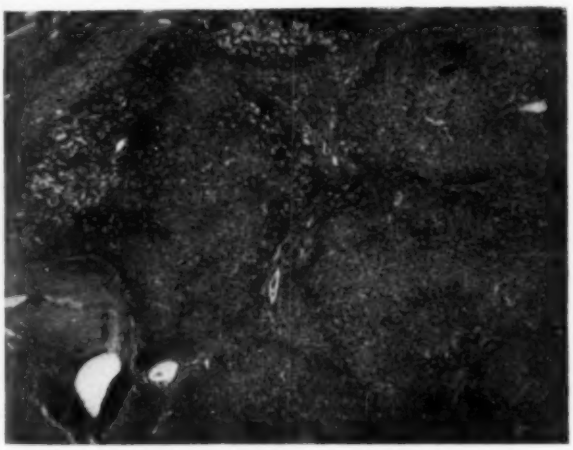

FIG. 691.—Photomicrograph showing varying sized lobules of parenchymal liver cells.

AT OPERATION.—A thin-walled gall-bladder, containing multiple small white stones, was removed and the common bile-duct explored. Here, numerous stones of a similar type were washed out and no further abnormalities of the common bile-duct, pancreas, or duodenum were encountered. The remaining abdominal organs, other than the liver, were normal. On the inferior surface of the liver was a tumour situated in the left lobe just medial to the gall-bladder and above the extrahepatic portions of the hepatic ducts. This tumour formed a smooth nodule measuring 4-5 cm. in diameter, and was firm and a homogeneous dark red colour. Large veins coursed over its surface, which was raised from the surrounding liver tissue by one centimetre. It was round and completely demarcated.

The tumour did not have the umbilicated surface of a typical carcinomatous metastasis and was considerably firmer than a hæmangioma. It did not appear to be arising from any part of the biliary tract and it was thought that the most likely diagnosis was hepatoma. The tumour was then enucleated and displayed a firm

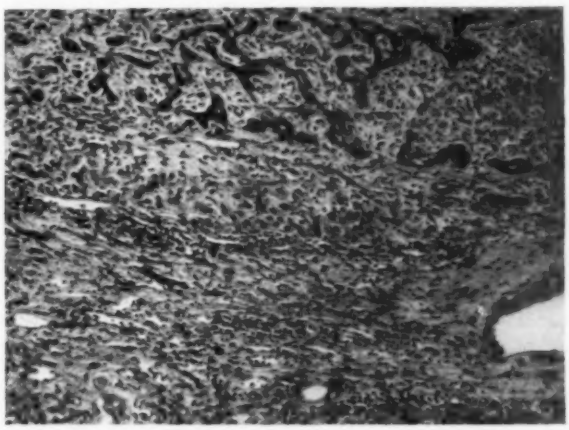

FIG. 690.—Photomicrograph showing dense lymphocytic infiltration.

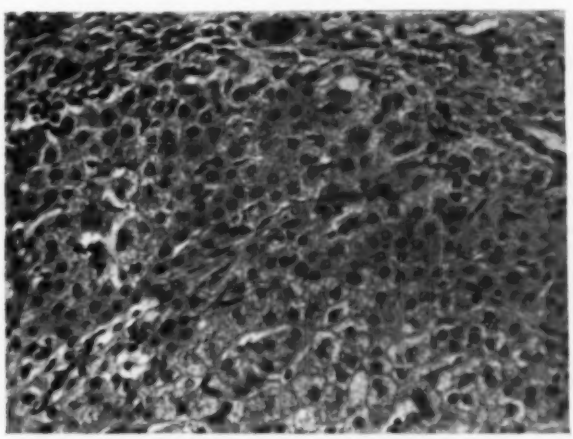

FIG. 692.—Photomicrograph showing cells at periphery, smaller, more deeply stained, and closely packed.

capsule surrounding it completely. The patient's post-operative course was uneventful.

PATHOLOGY.—The specimen (*Fig.* 689), measuring 4·8 cm. in diameter, was bosselated and covered by a thin layer of connective tissue suggestive of a capsule. Its outer aspect, where it projected above the surrounding liver tissue, was smooth. The deep aspect was essentially smooth, with a few fibrous tags representing the attachment to its bed in the liver. Slight linear thickenings in the 'capsule' were irregularly distributed over the surface, lying chiefly in the depressions between the bosses. The nodule appeared complete and intact.

The cut surface revealed a central stellate fibrous core from which fibrous septa extended peripherally to divide the tissue into small lobules of varying size. This became more apparent following fixation. The tissue between the whitish fibrous septa was tawny brown in colour and bulged slightly above the fibrous component of the nodule. The consistency was firm, with no evidence of hæmorrhage, degeneration, or necrosis.

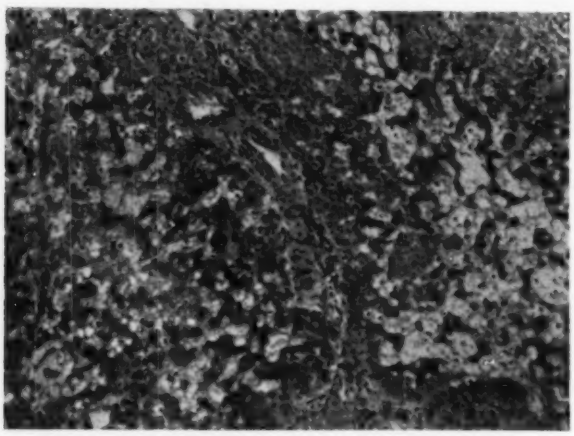

FIG. 693.—Photomicrograph showing hydropic degeneration of parenchymal cells in chequer-board fashion.

Microscopically, the limiting membrane was incomplete and consisted at most of two or three parallel collagen fibres. Where these were lacking, the periphery was formed by compressed liver cells. The central stellate core and its radial ramifications were composed of loosely textured fibrous tissue. This contained many thin-walled vascular channels and, in addition, narrow cords of parenchymal liver cells, proliferating bile-ducts, and a lymphocytic infiltration which, in some areas, was dense (*Fig.* 690). The bulk of the lesion, however, was formed by varying sized lobules of parenchymal liver cells (*Fig.* 691). Within individual lobules the central cells were large, pale, and possessed abundant vacuolated cytoplasm. The cells at the periphery tended to be smaller, more deeply stained, and more closely packed, suggestive of more active cell growth (*Fig.* 692). Some of the lobules revealed hydropic degeneration of parenchymal cells in a somewhat chequer-board fashion (*Fig.* 693).

Thin-walled central veins showed no regular distribution, and some lobules contained two or three of these. Bile plugs were few in number and were found between parenchymal cells—not in identifiable bile canaliculi. Fatty metamorphosis was absent, as was alcoholic hyaline, described by Benz and Baggenstoss (1953) in a small percentage of cases of similar nature.

Because of the similarity of this lesion to the changes seen in portal cirrhosis involving the entire organ, a diagnosis of focal cirrhosis of the liver was made.

Case 2.—Mrs. H. M., aged 53. A 5-day history of increasing large bowel obstruction necessitated this patient's admission to hospital. Radiographs showed an obstructing lesion of the left colon, which subsequently proved to be an adenocarcinoma with local glandular involvement only. At the time of resection of this tumour a nodule on the edge of the tip of the left lobe of the liver was encountered. This nodule measured 1½ in. in diameter, and was raised, round, and well demarcated from the remaining liver, which was normal. The surface was nodular rather than smooth, and was brownish-pink in colour. Again, this lesion did not have the appearance of a secondary carcinoma, and a diagnosis of localized cirrhosis of the liver was made and a biopsy performed.

PATHOLOGY.—The liver biopsy specimen was a firm piece of light brown tissue measuring 1·5 cm. in diameter.

On cut surface it was finely lobulated, with fine fibrous septa separating lobules. The intervening tissue was pale brown and grey, flecked with yellow. The histologic features were similar to those of the preceding case (*see Fig.* 693). The bowel contained an adenocarcinoma, which had extended to regional lymph-nodes.

DISCUSSION

There are two aspects of these rare lesions which are of practical importance. The surgeon, mistaking such a nodule for metastatic carcinoma, may desist from performing a curative operative procedure. This problem is readily solved by a frozen section. Primary carcinoma of the liver, when present, is commonly associated with cirrhosis of the liver. The removal of localized cirrhotic nodules, when the opportunity presents, is, at least on theoretical grounds, perhaps worthy of consideration.

There is no significant age or sex distribution. Those nodules found in children tend to be larger and, despite their benign appearance, grow more rapidly. In most instances a solitary nodule is found either incidentally at operation or at autopsy. Symptoms, when present, are usually resultant from the presence of a mass, with associated pressure phenomena. While it may be anticipated that other abnormalities in the liver or biliary tree and gall-bladder would be a common accompaniment, such is not the case. The miscellany of conditions with which it has been incidentally associated fails to suggest any constant causal relationship. Jaundice is very rarely present and, when found, is of extra-hepatic origin.

While focal cirrhosis of the liver usually occurs as a solitary lesion, rare instances of two or three nodules in one liver have been reported (Benz and Baggenstoss, 1953). Varying from a few millimetres to many centimetres in diameter, most lesions are sharply demarcated. The demarcation is due either to condensation of fibrous tissue or compression of parenchymal cells at the periphery of the nodule. There is no true capsule. The commonest location is in the right lobe of the liver, particularly its inferior surface, where it may project above the surface, be flush with the surface, or be surrounded by a moat-like depression at its periphery. While many of the lesions are subcapsular, they also occur deep in the hepatic parenchyma.

As a review of the literature clearly illustrates, there is no universally acceptable theory of pathogenesis. What could illustrate this better than the multiplicity of names under which these nodules have been described? Adenoma, benign hepatoma, hamartoma, solitary hyperplastic nodule, and focal

cirrhosis are all terms used by various authors to designate the same pathological entity. Here represented are neoplastic, congenital, and reparative (inflammatory) causations. Since, histologically, as well as in its gross appearance, the lesion so perfectly duplicates the features of portal cirrhosis, we prefer the designation ' focal cirrhosis of the liver ', thus accepting a pathogenesis based on a response to injury.

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APPENDICO-VESICAL FISTULA

REPORT OF A CASE AND SURVEY OF THE LITERATURE

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THE subject of appendico-vesical fistula has received but scant mention in the literature, although individual cases have been reported from time to time, and brief references to the subject have also appeared in articles on the broader subject of vesico-intestinal fistula in general. During the present century at least four authors have surveyed the world's literature on vesico-intestinal fistula, and are usually quoted in any discussion on the subject. They are Pascal (1900) 282 cases; Parham and Hume (1909) 385 cases; Albano (1926) 438 cases; and Kellogg (1938) 592 cases (to which he added one personal case), the largest series in the literature. Kellogg, however, omits to mention 54 cases (35 quoted by Higgins (1936) and 19 by Brasch in a discussion of Higgins's paper), but these 54 are included in a later series of 148 cases published in 1954 by Williams, who examined the literature for the previous twenty years. As only 6 of Williams's cases occurred early enough to be included in Kellogg's series, the total number of vesico-intestinal fistulæ up to and including Williams's paper is therefore 593, plus 142, i.e., 735.

Of these 735 cases, 31 were examples of appendico-vesical fistula, but some cases have been missed by the above authors and require to be added to this list. Thus Mackenna, in the discussion of Higgins's paper, mentioned 3 cases not included in the above list, and detailed descriptions of cases have also been given by Pool (1940), Keen (1898), Goldstein (1938), Rankin and Judd (1921, 2 cases), and Meisel, Kagan, and Miller (1949), making a total of 40 cases, to which I wish to add a personal case.

CASE REPORT

A male patient, aged 39, was first admitted to hospital on Aug. 21, 1949, with a diagnosis of ' ? acute appendicitis '. His illness had begun on the previous day with sudden griping pain in the right lower abdomen, colicky in nature, coming in waves and making him roll around in an effort to ease it. It was situated just below McBurney's point, with radiation to the penis and right testicle, and was accompanied by profuse sweating and vomiting. He had three mild attacks during the night before admission, and a severe one on the day of admission. White-cell count was 21,000 per c.mm. and urine examination showed a few pus cells. His temperature, pulse, and respiration rate were all normal. The pain soon settled and a provisional diagnosis was made of ' ? right renal colic, ? acute right pyelonephritis '. He was given a course of sulphamerazine, with resolution of his symptoms, and 4 days after admission he was discharged. A subsequent intravenous pyelogram (Sept. 21, 1949) revealed a normal urinary tract with no sign of calculus.

Following his discharge from hospital he passed " about a pint of pus and blood " per urethram and was cystoscoped, but owing to bleeding a satisfactory view

FIG. 694.—Rough sketch of operative findings. 1, Appendix; 2, Small bowel adherent to bladder; 3, Bladder-wall; 4, Prolapsed portion of appendix protruding into bladder.

was not obtained. His acute symptoms subsided with the help of aureomycin, but his urine remained infected for years, and he also suffered from occasional bouts of swelling of the right testicle. Eventually, he was again cystoscoped, but bleeding again obscured the view, and some two months after this last examination he was admitted to hospital (Sept. 25, 1956) where I saw him for the first time. His total non-protein nitrogen was 33 mg./100 c.c., hæmoglobin 15·6 g./100 c.c., and his urine contained a heavy growth of *Bacillus coli*, sensitive to streptomycin, terramycin, and chloromycetin. Cystoscopy under anæsthesia (Sept. 27) revealed only a very mild degree of cystitis, but situated on the right posterior wall of the bladder, high up near the fundus was a small, finger-like, solid-looking nodule with a small indentation at its tip. It had the appearance of a prolapsed ureter, but the normal ureteric orifice could be seen below it. A biopsy was taken and this was reported on as " mucosa of large bowel or vermiform appendix ".

AT OPERATION (Oct. 1).—A laparotomy was performed through a lower midline incision, and the bladder was also opened, the small nodule already described being easily seen projecting into it. The operative findings are shown diagrammatically in *Fig.* 694. The small bowel was dissected from the bladder, and a partial cystectomy performed by excising a piece of the thickened and indurated bladder-wall surrounding the nodule already mentioned. The appendix was now able to be delivered with ease into the wound, and was found to be grossly thickened, almost to the size of one's thumb.

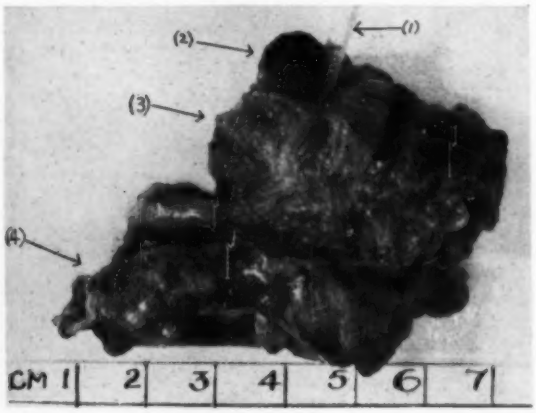

FIG. 695.—The specimen showing 1, Probe in fistula; 2, Prolapsed portion of appendix; 3, Thickened and indurated bladder-wall; 4, Appendix.

Appendicectomy was performed, and the bladder sutured and drained by a suprapubic catheter. Complete closure with urethral catheter drainage was considered but rejected as a fairly large piece of bladder-wall had to be excised and the capacity of the organ thereby reduced.

The patient's post-operative condition was good, and on Oct. 7 the suprapubic catheter was removed and a urethral catheter passed into the bladder. The suprapubic wound healed gradually, and on Oct. 22 the urethral catheter was removed, and as the patient was able to void well without suprapubic leakage, he was discharged from hospital the next day.

PATHOLOGY.—

Macroscopic.—A thickened portion of bladder-wall approximately 2 cm. thick, with mucosal surface 2 cm. × 3·5 cm. Appendix attached externally by dense fibrous adhesions, the appendix measuring 5 cm. in length, and approximately 1·5 cm. in thickness. Part of the meso-appendix is attached. Near one edge of the bladder mucosa there is a papillary projection covered by congested mucosa and measuring 1·5 cm. in height and 0·9 cm. in diameter. At its base is a small opening through which a probe has been passed for 2·5 cm. through the wall towards the attached appendix (*Fig.* 695).

Histology.—A portion of the wall of the appendix near the base shows a normal mucous membrane with a few lymphoid follicles. In the submucosa there are scattered eosinophils throughout the muscle layers. There are engorged blood-vessels on the peritoneal coat. The appearances are those of a chronic appendicitis.

A section from the portion of appendix inside the bladder shows mucous membrane with a few muscle fibres. These appearances suggest a prolapse of the mucous membrane of the appendix into the bladder.

Four months post-operatively (Feb. 8, 1957) the patient stated that he had made a complete recovery and felt " better than for years ".

SURVEY OF THE LITERATURE

I have been able to find detailed reports of 11 other cases of appendico-vesical fistula in a search of the literature of the past 35 years. A brief résumé of these cases is given below.

1. Dobson and Moir (1932).—Male, aged 19. Abdominal pain, temperature 102°. Diagnosis of appendicitis considered but rejected. Later discharged 3 oz. pus per urethram, mass right side abdomen discovered. Laparotomy 5 weeks after onset of original pain. Perforated appendix, small abscess cavity opening into bladder. Appendicectomy, suprapubic drainage (bladder-wall indurated and suturing therefore abandoned). Recovery.

2. Pool (1940).—Male, aged 36. Symptoms predominantly urological. Cloudy urine 4 years, frequency, dysuria, hæmaturia. I.V.P.—hour-glass shadow in region of bladder. Cystoscopy—vesical calculus. Litholapaxy. Six days later fæcal material in urine. Barium enema, cystogram both normal. Cystoscopy—small opening right side of bladder, ureteric catheter inserted, opaque medium injected, right half of colon filled. Appendicectomy and urethral catheter drainage. Recovery.

3, 4. Rankin and Judd (1921).—*a.* Male, aged 36. Symptoms predominantly urological—frequency and burning 7 years. Three cystoscopies in 6 years: (1) " Diverticulum and enlarged prostate ". (2) " Enlarged prostate ". (3) " Ulcerative cystitis dome of bladder, and chronic prostatitis ". Radiograph of urinary tract negative. Laparotomy, freeing adhesions from last 18 in. small bowel, peritonealizing raw surface, Witzel enterostomy, suprapubic cystostomy (presumably also appendicectomy though authors do not actually say so). Recovery. *b.* Male, aged 33. Pneumaturia, 8 years, also pus, gas, fig-seeds, food per urethram. ? Acute appendicitis prior to first attacks of passage of gas. Radiography of urinary tract negative. Barium enema—fistula cæcum to bladder. Cystoscopy—intestino-vesical fistula (*see* Discussion). Appendicectomy, closure of bladder. Recovery.

5. Keen (1898).—Quoted by Lower and Farrell (1931). Male, aged 24. Dysuria since age of 7 when family doctor removed pin from urethra. Child denied having inserted one, could not remember swallowing one. At 23, " recto-vesical fistula secondary to rupture of prostatic abscess " diagnosed; two unsuccessful attempts at closure. One year later, laparotomy, long appendix, tip incorporated in bladder. Appendicectomy. Recovery.

6. Lower and Farrell (1931).—Male, aged 26. Past history " bladder stones ". Cystoscopy twice—both negative. In 1925, fell downstairs and " jarred a stone loose from bladder wall ". Radiography showed stone surrounding a pin. Removed from bladder 1 week later (swallowed at age of 6 months). After this, recurrent stones removed 1926, 1927. In 1950 hæmaturia, pain right kidney, fæcal-looking material in urine. I.V.P.—slight right hydronephrosis. Cystoscopy—gross cystitis, fæcal material in bladder, depression near top of bladder suggesting fistula connected with bowel. Appendicectomy. Invagination of bladder wall. Recovery.

7. Goldstein (1938).—Male, aged 59. Painless hæmaturia 2 months. Previous history, attacks of constipation, abdominal discomfort. Cystoscopy—tumour left posterior lateral wall of bladder and benign prostatic hypertrophy. Tumour fulgurated on several occasions; severe reaction after each one, severe abdominal pain after one of them. Fulguration, radium implantation, and transurethral resection decided upon. On further bladder examination small elevated area, with slight depression in centre, seen on right posterior lateral wall, size of a large pea. Thought to be another tumour; fulgurated. Immediate severe pain, pallor, collapse, weak and rapid pulse. Gradual deterioration. Sixty hours post-operatively abdomen opened—fibrino-purulent exudate, appendix adherent to bladder, ileum accidentally perforated by surgeon. Procedure: Ileostomy, drain to pelvis, catheter to bladder. Patient died

9 hours later. Autopsy: Pin-point perforation in bladder-wall alongside appendix, bladder-wall very thin here (1 mm. thickness at thinnest point). Cause of death—peritonitis, bronchopneumonia. (This case has been described in some detail as the cystoscopic appearance of the appendix was similar to my own case. A discussion will follow later.)

8. **Meisel, Kagan, and Miller (1949).**—Male, aged 25. Intermittent lower abdominal pain, diarrhœa 3 weeks, frequency, terminal hæmaturia, 2 weeks. Pneumaturia 1 week. Attack of lower abdominal pain 4 years previously. Cystoscopy—acute cystitis and large soft calculus, small area of ulceration right side of bladder. Barium enema negative, but barium later passed in urine. Appendicectomy and removal of small cuff of bladder. Bladder suture, indwelling catheter. Recovery.

9. **Howe (1932).**—Female, aged 19. Pain right side abdomen, nausea, frequency, burning several days. Temperature, pulse, respiration all normal. Tenderness and rigidity in right iliac fossa. White-cell count 10,000. Urine—pus and cellular debris. (Similar attacks over previous 12 months.) Laparotomy for "surgical abdomen". Appendix adherent to bladder, appendicectomy, closure of bladder, drain to peritoneal cavity. Recovery.

10, 11. **Nesbitt and Coller (1934).**—*a*. Female, aged 13. Diarrhœa, lower abdominal pain. Burning on micturition 10 years, worse last 3 years. Ten years before, stone removed from bladder with pin embedded in it, but symptoms persisted and second operation on bladder (nature not stated) unsuccessful. Cystoscopy—3 depressions right side of bladder, could not catheterize any of them. Bladder filled with fluid which later passed per rectum, but nothing felt on examining finger. Operation, appendicectomy, tip buried in bladder-wall. Closure of bladder, urethral catheter, 12 days. Discharged 14th day. Recovery.

b. Male, aged 33. Symptoms mainly urological, 2 days' severe dysuria, burning in urethra, frequency, pneumaturia, gross fæcal material in urine, diarrhœa 1 day. Past history: At age 10 painful and burning micturition which continued on and off 10 years. Since then infrequent attacks of dysuria, occasional chills, fever, right costovertebral tenderness. Urological investigation: "No tuberculosis, no infection upper urological tract, chronic prostatitis and seminal vesiculitis." Passed small stone at age 32. At 33, findings were fistulous opening on floor of bladder medial to right ureteric orifice, no attempt to catheterize because of discomfort to patient. I.V.P. and cystogram both negative. Barium enema—appendico-vesical fistula. Operation—long appendix adherent to bladder; appendico-vesical fistula. Appendicectomy, bladder drained suprapubically. Recovery.

Apart from the 11 cases quoted above, other cases have been briefly mentioned in the literature, as described in the opening sentence of this paper. Most of these are quoted briefly in the larger series of cases of vesico-intestinal fistula, and often include only the barest details, e.g., age, sex of the patient, type of operation, result, and little else. In some cases even less information is given and few conclusions can be drawn. The following discussion of the subject is therefore based mainly on the 12 cases described in detail, implemented by such brief information as is available about the other cases quoted.

DISCUSSION

Incidence.—Of the combined series of Kellogg and Williams (735 cases of vesico-intestinal fistula) 31 were examples of appendico-vesical fistula, i.e., 4·3 per cent. This figure is probably fairly accurate, as although some cases of appendico-vesical fistula have not been included in the total given, this would be offset by the fact that there would be a greater tendency to report single cases of appendico-vesical fistula (because of their rarity) than of most of the other types of vesico-intestinal fistulæ. In the main the percentage figures given by various authors have agreed with the figure quoted above, e.g., Pascal 3·3 per cent, Albano 8·2 per cent, Kellogg's personal series 3·4 per cent. Sutton (1921), in the largest series from one institution, viz., 59 cases, found 3 cases of appendico-vesical fistula, i.e., 5·04 per cent.

It is convenient at this point to consider briefly why, when acute appendicitis is so common, appendico-vesical fistula is so rare, especially when we consider that acute diverticulitis, which is much rarer than acute appendicitis, is a much commoner cause of fistula.

The answer lies mainly in the fact that whereas in acute appendicitis the usual treatment is early surgery before fistula formation has a chance to occur, in acute diverticulitis conservative management is the rule, and this leads to the possibility of a fistula occurring in course of time. As will be shown later, the cases of acute appendicitis which give rise to appendico-vesical fistula are also those in which conservative treatment has been undertaken, either because of the trivial nature of the symptoms or because of mistaken diagnosis.

The sex incidence of vesico-intestinal fistula in general shows a marked predominance for males, mainly due to the interposition of the uterus and female adnexa between the bladder and bowel, and to the fact that diverticulitis is commoner in the male than in the female. Thus the percentage of males affected in the series of cases of vesico-intestinal fistula quoted by different authors has been as follows: Pascal 75 per cent, Albano 67·11 per cent, Kellogg 67·6 per cent, Williams 75 per cent. The same predominance of males is noted in appendico-vesical fistula, and is even more pronounced. I have been able to find 23 cases in which the sex is mentioned, and of these 19 (82·6 per cent) are males.

The age incidence of acute appendicitis being lower than that of either carcinoma or diverticulitis, we would naturally expect this to be reflected in the age incidence of appendico-vesical fistula as compared with vesico-intestinal fistula in general, and this is borne out by such figures as are available. I was able to find 21 cases of appendico-vesical fistula in which the age was mentioned, and although it varied from 7 to 62, 16 of the cases (76·2 per cent) occurred between the ages of 10 and 40 (10–20, 4 cases; 20–30, 5 cases; 30–40, 7 cases). These figures contrast with those of Kellogg for vesico-intestinal fistula in general, his findings being 37 out of 55 cases (67·3 per cent) in the 40–70 age-group.

Pathology.—Theoretically the lesion causing an appendico-vesical fistula may arise either in the bladder or the appendix. I can find no record of any bladder lesion, such as a neoplasm, having ever ulcerated through to the appendix (and indeed such an occurrence must be excessively rare), the causative lesion always having been in the appendix. In the vast majority of cases this has been an acutely inflamed appendix which is pelvic in position and

as a rule longer than usual, occupying a position in close proximity to the bladder. The inflamed appendix then becomes adherent to the bladder causing secondary inflammation of its wall. As the inflammation progresses to the stage of abscess formation, rupture through into the bladder occurs. It is worth emphasizing that adhesion of the appendix to the bladder precedes fistula formation. The fistula is not simply the result of perforation of a (pelvic) appendix abscess into the bladder, as such an abscess is much more likely to burst into the rectum than into a thick-walled viscus such as the bladder. Thus every detailed account has described the tip of the appendix as being adherent to the bladder-wall, often requiring to be dissected from it. The position of the fistula is also fairly constant, being always on the right side of the bladder, usually high up near the fundus opposite the appendix, but of course varying from case to case. In one case (Nesbit and Coller) it was described as being on the base of the bladder, the appendix in this case being 7 cm. in length. Adhesion of the tip of the appendix to the bladder will not, of course, lead to fistula formation, unless such adhesion is followed by abscess formation and perforation as already described. Perforation into the bladder will however be assisted if a sharp foreign body, such as a pin, is present within the appendix, and it is significant that a pin is mentioned in no fewer than three of the 11 cases quoted above (*Cases* 5, 6, 10).*

Other lesions of the appendix (apart from acute inflammation) may give rise to a fistula, but are rare. Mackenna mentions 2 cases in which tubercle bacilli were found in the urine, but ureteric catheterization was negative. His account, however, does not make it clear if the lesion causing the fistula was tuberculosis of the appendix, or acute appendicitis occurring in a patient with tuberculosis of some other part of the bowel (e.g., the cæcum). Actinomycosis of the appendix has been cited as a cause in 2 cases, one being quoted by Parham and Hume, and another one later by Albano, but no details of these cases are given. Tumour of the appendix also springs to mind as a possible cause of these fistulæ, but I have not been able to find any record of such a case.

The changes in the bladder-wall in appendico-vesical fistula have followed what one expects in such a lesion. Induration at the site of fistula formation has been mentioned by more than one author and was well marked in my own case. On two occasions (Goldstein's case and my own) prolapse of the appendiceal mucosa through the fistula into the lumen of the bladder has occurred. The remainder of the bladder-wall has shown evidence of cystitis in a greater or lesser degree, and in general where the opening into the bladder is comparatively large, not only is the degree of cystitis greater, but it is in these cases that one finds gas, food, or fæces in the urine either separately or in combination. These facts are, of course, self-evident and apply to all types of vesico-intestinal fistulæ. In the 11 cases of appendico-vesical fistula however, gross evidence of fæcal contamination was present in 5 cases only, as blockage of a long thin tube such as the appendix can readily occur. Thus in my own, and in Goldstein's case, where prolapse of the mucosa had occurred and blocked the opening, evidence of cystitis was minimal and there was no sign of fæcal contamination of the urine. On the other hand, in Rankin and Judd's second case, where the fistulous opening was comparatively wide and easily observed both at cystoscopy and by barium enema, fæcal contamination of the urine was well marked and both gas and food particles were present in the lumen of the bladder.

Stone formation in the bladder is mentioned in 5 of the 12 cases, infection and the presence of a foreign body being the two main aetiological factors. Thus in both cases where a pin was found in the bladder it was surrounded by a stone, and of the 5 cases where fæcal contamination of the urine was present in one form or another, 4 were complicated by the presence of a bladder stone. Although detailed descriptions of these stones are not given it is possible and indeed probable that they may have arisen round a nucleus consisting of a foreign body such as a small piece of food, a seed, or a fæcoalith, and that their continued growth was aided by the gross fæcal contamination present. Of the 6 cases where *either* fæcal contamination *or* foreign body in the bladder was mentioned, only one was free from vesical calculus (*Case* 4). One cannot, of course, draw too many conclusions from such a short series of cases but the above facts are, to say the least, suggestive.

Clinical Features.—The symptoms of appendico-vesical fistula are so variable that it is difficult to assess what is the typical clinical picture.

On first thoughts one might expect to find the usual symptoms of acute appendicitis, followed by those of a fistula to the bladder with fæcal contamination of the urine. A moment's reflection, however, will reveal the fallacy of this reasoning. In the first place an attack of acute appendicitis with typical symptoms will usually lead to early appendicectomy before there is a chance for fistula formation to occur. In the second place, nowhere else in the bowel is there a long thin tube analogous to the appendix, with such possibilities of blockage of the lumen after fistula formation has occurred. Gross fæcal contamination of the urine is thus by no means an invariable feature, as has already been pointed out. The symptoms are therefore liable to be confusing in both the early and the later stages of the condition, and uncertainty and delay in diagnosis is the rule rather than the exception. The average length of time between the initial symptoms and the final diagnosis in the 11 cases quoted could thus be measured in years, and only in one case was this time interval reasonably short (*Case* 1), where the presence of a mass led to surgical exploration within five weeks of the onset of symptoms. In general the vagueness of the history together with the rarity of the lesion makes diagnosis very difficult indeed. Out of the chaotic collection of symptoms which have been recorded in appendico-vesical fistula we may therefore summarize the main characteristics of the clinical picture as follows: (1) Abdominal symptoms which are vague, are not typical of acute appendicitis, and are liable to be overlooked or disregarded;

* In one of these three cases, however, aetiological relationship of the pin to the fistula is open to doubt (Keen's case). The child may have inserted it into his urethra.

(2) Urinary symptoms varying from those of a mild infection to those of a marked cystitis with fæcal contamination of the urine, with or without the symptoms of a vesical calculus; (3) Owing to the unobtrusiveness of the symptoms, chronicity is the rule, the history often being measured in years.

In most cases, the initial symptoms having been disregarded, the case will present itself as one of chronic urinary infection requiring investigation by urine culture—including a search for tubercle bacilli—intravenous pyelography, cystoscopy, etc. If fæcal contamination of the urine is present, a vesico-intestinal fistula will at once be suspected, but otherwise a gradual process of elimination will be necessary. Diagnosis will in most of these cases depend mainly on the results of cystoscopy and the barium enema.

At cystoscopy it is evident from the published reports already quoted that a fistulous opening has not usually been discovered unless it was already suspected and a specific search made for it. Thus in Rankin and Judd's first case (in which there is no mention of fæcal contamination of the urine) three cystoscopies were performed and in none was the fistula discovered. On the other hand, in their second case (in which gas, pus, and food were all present in the urine) the fistula was observed at once on cystoscopic examination. This is probably mainly due to a specific search being made for it, although as already pointed out, in the case with fæcal contamination of the urine, the fistulous opening is likely to be wider and thus more easily seen.

Even though a search is made for a fistula, in some cases it may be hidden by a large bladder stone, and this may have been a factor in Pool's case, in which the fistula was missed at the first cystoscopy (when a vesical calculus was present), but seen later after litholapaxy had been performed. However, as fæcal matter in the urine was observed prior to the second cystoscopy, but not to the first, this may have been the main factor in assisting the diagnosis simply by promoting awareness of the presence of a fistula. If a fistulous opening is observed but its site of origin from the bowel is uncertain, a ureteric catheter should be gently passed into it, an opaque medium injected and a radiograph taken. Such an investigation may be of extreme value, as in Pool's case where barium enema was reported as negative, but after catheterization of the fistula and injection of the opaque medium, the right half of the colon was observed to be filled.

Prolapse of the tip of the appendix through the fistula into the bladder deserves a brief mention, as this has occurred in 2 cases (Goldstein's and my own). In both cases the appearance was suggestive, but not typical of a bladder tumour, the presence of genuine tumours elsewhere in the bladder in Goldstein's case rendering the diagnosis especially difficult. A word of warning is necessary regarding biopsy of such a prolapse. In my own case it was performed quite safely, but in Goldstein's case the mass was fulgurated and this resulted in a fatal perforation of the bladder. It is as well, therefore, to realize that the bladder-wall may be abnormally thin at the site of such a prolapse, and biopsy must be conducted with care and an awareness of the dangers present. Finally, the remainder of the bladder may show little sign of inflammation, or may be the site of a generalized cystitis according to the degree of infection present. This has already been considered in the discussion on pathology.

Barium enema may reveal much or little. In Pool's case (where the fistula was small) the barium enema was reported as normal. In Meisel, Kagan, and Miller's case it was also reported as 'negative', but barium was later recovered from the urine, proving a fistula, but giving no information about its site. Rankin and Judd's second case provides what is probably the most interesting barium enema of this series. The colon was observed to fill as far as the hepatic flexure, but the barium could not be forced into the cæcum, which was noted to be filled with fluid. The patient was then asked to void urine while being screened and the barium was seen to enter the cæcum (this urine specimen was free from barium). On further observation barium was observed to flow through the appendix into the bladder, and a voided specimen of urine was now found to contain barium. A confident diagnosis of appendico-vesical fistula was therefore made.

It is clear from the above brief survey of the clinical features that diagnosis is extremely difficult especially where no fæcal contamination of the urine has occurred. In such cases awareness of the condition, as in so many other rare lesions, will be the main factor in making a pre-operative diagnosis.

Treatment.—Appendico-vesical fistula is the most satisfactory of all the bladder-to-bowel fistulæ with which we have to deal. Appendicectomy is of course the first step. It is almost superfluous to add that the ease or difficulty of this will vary considerably from case to case. Full operative details have not been given by every author, but as abscess formation precedes fistula formation adhesions, will be present requiring division, and as the distal end of the appendix is adherent to the bladder-wall it will require mobilization. In some cases it may even be necessary to remove a cuff of bladder round the site of the fistula. This was necessary in my own case, for example, where prolapse of the appendiceal mucosa into the bladder had occurred. By and large one should be prepared to encounter a certain amount of difficulty in the removal of the appendix. The second step is suture of the bladder-wall. It is usually possible to close the bladder completely, with urethral catheter drainage, but if the wall is indurated or for some other reason suture is difficult, it is safer to drain the bladder by a suprapubic catheter. A drain to the cavity of Retzius should never be omitted.

Prognosis.—From the viewpoint of prognosis also, the condition may be described as the most satisfactory of this group of fistulæ. Whereas the mortality of vesico-intestinal fistulæ in general is high (46·6 per cent in Kellogg's series) the mortality of a well-conducted operation for appendico-vesical fistula is very low. Of the 11 cases quoted in this paper appendicectomy (usually combined with suture of the bladder as described above) was performed in 11 cases with 100 per cent recovery. The one death (Goldstein's case) did not result from appendicectomy, but occurred following a most unusual accident, as has already been discussed.

The foregoing excellent results are in marked contrast with those of half a century ago. In 1909, for example, Parham and Hume quoted 9 cases of appendico-vesical fistula, and of these 9, no fewer than 7 died. Details of the treatment given are very scanty, but in 5 cases (all fatal) there is no record of an operation having been performed. In short, modern surgery has completely altered the prognosis of what was once a very dangerous condition, and nowadays it may be truly said that if one has to have a fistula from bowel to bladder, then an appendico-vesical fistula is the best one to choose.

SUMMARY

1. A case of appendico-vesical fistula is reported.

2. Appendico-vesical fistula is rare, about 40 cases having been mentioned in the literature. It probably represents about 4–5 per cent of all vesico-intestinal fistulæ.

3. About 4 out of every 5 cases occur in the male sex. Three-quarters of the cases occur between the ages of 10 and 40. The age-incidence of vesico-intestinal fistula in general is higher, two-thirds of the cases being in the 40–70 age-group. This is because the age-incidence of carcinoma and diverticulitis of the bowel is higher than that of acute appendicitis.

4. The causative lesion is usually acute appendicitis, with adhesion of the distal end of the organ to the bladder-wall, abscess formation, and rupture through into the bladder. Other inflammatory lesions of the appendix such as tuberculosis or actinomycosis are only rarely the cause, and tumour of the appendix as a cause of the fistula has not been reported. A foreign body (such as a pin) in the appendix may assist fistula formation. Fæcal urinary contamination occurs only in about half the cases, as the long thin lumen of the appendix lends itself to blockage after the fistula has occurred. Vesical calculus has been reported in about 40–50 per cent of the cases and is more likely to occur if a foreign body is present, or if fæcal urinary contamination occurs. Prolapse of the appendiceal mucosa through into the bladder may occur, the cystoscopic appearances of such a prolapse being very suggestive of neoplasm.

5. The symptoms of appendico-vesical fistula are not constant, but the main characteristics of the clinical pictures are : (1) Vague abdominal pains, not typical of acute appendicitis, liable to be overlooked, followed by (2) Urinary symptoms, chronic urinary infection varying in severity according to the degree of fæcal contamination of the urine.

6. Diagnosis depends mainly on the cystoscopic findings and the barium enema, but the fistula is by no means always seen at cystoscopy nor is it always visualized in a barium enema examination.

7. The treatment is operative and consists of appendicectomy and suture of the bladder, which is usually drained by urethral catheter. If suture of the bladder wall is unsatisfactory for any reason, drainage is better effected by suprapubic catheter. The cavity of Retzius should always be drained.

8. The prognosis following operation is excellent, and only 1 death has been recorded in a series of 11 cases. No other type of vesico-intestinal fistula has such a good prognosis.

I would like to record my thanks to Mrs. H. K. Cutten, of the Central Medical Library, Auckland, for her valuable help in searching the literature ; to Dr. N. A. Bennetton of the Modern Languages Department of Auckland University College for translating for me the article by Albano ; and to my colleague Mr. E. Gifford for many helpful criticisms and suggestions.

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WRY MOUTH AFTER EXCISION OF THE SUBMAXILLARY GLAND

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In 1951 Hayes Martin, del Valle, Ehrlich, and Cahan in an article on block dissection of the cervical lymph-glands in carcinoma, drew attention to a branch of the facial nerve which supplied the ipsilateral lower lip, namely the ramus marginalis mandibulæ. They pointed out that unless identified, it was liable to be cut and a deformity of the lower lip produced. They considered that the danger point was where the nerve turned upwards over the edge of the mandible and came to lie superficial to the facial vessels, and advised identification of the nerve at this point when making the upper flap, using the upper ligature on the facial vessels to retract the nerve upwards out of harm's way.

In operations on the submaxillary gland, standard text-books do not suggest identification of the ramus marginalis mandibulæ, or mandibular branch of the facial nerve as it is more commonly known, as a definite step ; nor do surgeons with whom we have discussed the matter. From Hayes Martin's experience in cervical block dissection we concluded, that permanent damage to this small nerve would occur in removal of the submaxillary gland with equal facility. Since reading the article it has been our habit to identify and preserve the nerve in operations on the submaxillary gland. We have, as yet, not performed the operation sufficiently often to give final results, but our early impressions are favourable. Our interest in the problem has been aroused and this article is presented to record the following :—

1. The final deformity in a series of cases of submaxillary gland removal where no attempt to identify the nerve had been made.

2. A study of the anatomy of the mandibular branch of the facial nerve.

POST-OPERATIVE FOLLOW-UP

A consecutive series of 24 patients who had undergone removal of the submaxillary gland for calculous disease more than two years previously, was personally examined and the records inspected. Any temporary weakness by this time would have recovered. In none had any attempt been made to identify the nerve, so that in all cases it was potentially in danger. Out of these 24 cases, 15 were found to have a residual weakness. In none was anything noted with the mouth in repose, but in 7 the weakness was noticeable on smiling and in 8 on grimacing. Weakness immediately following operation was only recorded in 12 cases and in 3 of these complete recovery had occurred when examined by me. In assessing these cases I have looked for very minor changes ; even if the nerve is totally and permanently damaged the resulting deformity is slight and perhaps not noticed by the patient himself until pointed out to him by others. It has been suggested that a slightly crooked smile is not unattractive. In fact, none of the patients examined had any comment to make about their appearance ; nevertheless, it is clear that permanent, though mild and therefore partial, damage is done in an appreciable number of cases. The photographs show a patient soon after operation, firstly with the face in repose, and secondly smiling, revealing a typical wry mouth (*Fig.* 696).

ANATOMICAL STUDIES

To determine whether any operative manœuvre could prevent this damage, dissection of the nerve and submaxillary gland in 10 post-mortem subjects was carried out, providing 20 dissections. Simple line drawings were made at the time and perusal of these revealed the following facts :—

1. There was considerable variation in the size and shape of the submaxillary glands.

2. Bilateral asymmetry of all related structures was common.

3. There was considerable variation in the arrangement of the posterior auricular and posterior and anterior facial veins, resulting in variations in the common facial, external, and anterior jugular trunks.

Course of the Nerve.—

1. *Point of Emergence.*—In every dissection the mandibular branch of the facial nerve emerged from the lower pole of the parotid, either from the anterior border near the tip, or at the tip itself. It was always as a separate entity from the cervical branch, division of the lowest trunk having taken place inside the gland.

2. *Nerve Communications.*—No communicating nerves were found between the mandibular and cervical branches. In only one dissection was any connexion between the mandibular and a higher branch found, and this was a small twig passing across the masseter muscle from or to the lowest buccal branch.

3. *Relation to the Angle of the Jaw.*—After emerging from the parotid gland the nerve comes to lie in close relationship with the angle of the jaw. In 11 specimens the nerve passed behind but very close to the angle ; in 4 it passed behind and below the angle but never separated from it by more than 1 cm. ; in 3 it passed directly over the angle ; and in 2 it lay approximately ½ cm. in front of the angle. Some disturbance of relations inevitably occurred during the course of dissections and more exact measurements cannot be given.

This variation was due to the size and shape of the parotid gland and the site of emergence of the nerve. The farther the tip of the parotid descended over the sternomastoid muscle and the lower the nerve emerged from the gland, the more posterior to the angle of the jaw the nerve lay. Conversely, when the tip of the gland was short and the nerve emerged from the anterior border, it passed either directly over the angle or in front of it.

4. *Course in the Neck.*—The nerve then looped into the neck, the extent to which it descended varied, and it was noted that the farther the nerve lay behind the angle of the jaw the lower it descended into the neck ; however, it never lay more than 1 cm. below the jaw at any point. The ascending limb of the loop passed upwards behind, but quite close to

the posterior edge of the submaxillary gland to cross over the edge of the mandible immediately in front of the masseter muscle.

5. *Relation to the Facial Vessels.*—In every dissection the nerve came into direct relation to the facial vessels, in 5 just below the jaw, and in 15 just over the edge of the jaw. At this point it broke into two or three fine filaments.

A skin incision is made in a suitable skin crease. After division of the platysma its upper flap is dissected very carefully upwards off the deep fascia to the level of the mandible, revealing the deep fascia, the submaxillary gland, and facial vessels. The nerve is then identified, the facial vessels can now be doubly ligated close to the gland, and the upper ligature retained and pulled upwards—this

A B

FIG. 696.—Post-operative photographs after excision of the submaxillary gland. A, Face in repose; B, Smiling reveals a typical wry mouth.

Depth of the Nerve.—Passing through the parotid gland the nerve must lie deep to the deep fascia. After emerging from the parotid it retains this relationship throughout the whole of its course as exposed in operations on the submaxillary gland. The deep fascia in this region, however, is thinner and more areolar than other parts of the cervical fascia, but is definite enough, especially over the facial vessels, to provide protection from damage if dissection is carried out immediately deep to the platysma muscle.

CONCLUSIONS

These dissections show that the only fixed point of its course which lay within the standard operative field for removal of the submaxillary gland was just below or just on the mandible under the thin layer of deep fascia and superficial to the facial vessels. If not identified here, as advised by Hayes Martin in cervical block dissections, it is potentially in danger :—

1. At the point of ligature of the facial vessels above the gland.
2. If the incision is carried too deeply through the platysma to include the deep fascia.
3. When dissecting on a deeper plane behind the submaxillary gland, particularly if the nerve has looped into the neck.

Accepting the fact that the nerve is potentially in danger and that early identification is the only certain way of avoiding injury, the following technique for removal of the submaxillary gland is suggested.

retracts the nerve out of the operative field. The rest of the operation is carried out according to accepted methods.

Since this technique has been followed only a few cases have been treated; while temporary weakness has been noted, no permanent after effects have been encountered.

SUMMARY

1. Permanent weakness of the angle of the mouth may be produced after removal of the submaxillary gland; though the resulting deformity is slight it could be prevented.

2. Attention is drawn to the possible sites of damage to the mandibular branch of the facial nerve during removal of the submaxillary gland.

3. Early identification of the nerve is the only certain way of avoiding injury.

4. A simple technique is described for identification and preservation of the nerve.

Acknowledgements.—I wish to thank numerous consultant surgeons of the Middlesex Hospital for permission to follow their cases, and the Photographic Department for the two clinical photographs. I am particularly indebted to Mr. D. H. Patey for his helpful advice and criticism.

REFERENCE

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RECONSTRUCTION OF ARTERIAL CIRCULATION

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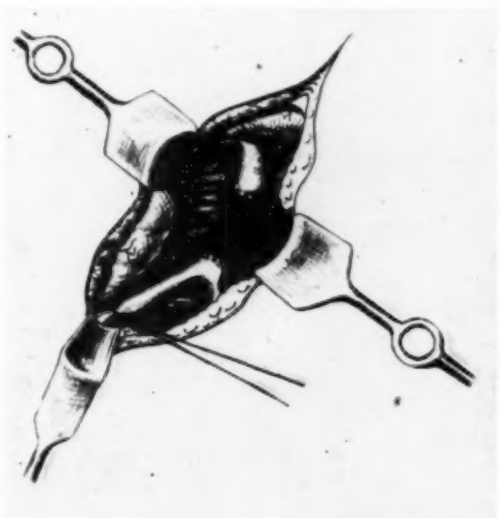
During the reconstruction of the peripheral arterial circulation complications have often to be faced, when even with the application of the best operative technique an initial thrombosis takes place which can make the operation unsuccessful.

The results of the repair of aortic coarctation are encouraging if the defect between the two resected ends is short, so that the anastomosis can be made without tension. In surgery of the peripheral arteries often defects amounting up to 30 cm. have to be overcome. For this purpose we use the near-by vein, if the lower extremity is involved, and we use this venous graft by turning it in the opposite direction and suturing it into the correct position. However, the results of these operations are not satisfying. After the graft has been sutured into the correct position we have observed that even half an hour later the circulation through the graft gets slower and the pulsation into the dilatated graft less visible, and disappears sometimes completely. In

Fig. 697.—Aortography. Spindle-form aneurysm of the left common iliac artery. Abdominal aorta is enlarged and extended.

A



B

C

Fig. 698.—A, Schematic view of the intra-operative condition. The peritoneum is displaced medially. The spindle-like aneurysm is shown lying between the bifurcation of the aorta and the branch of the hypogastric artery. B, Condition after resection of the aneurysm and blind occlusion of the arterial stumps. C, Reconstruction by bridging the defect with the hypogastric artery.

this case the operation is useless; there is a certain effect only which is due to the simultaneous arteriectomy.

The use of an arterial graft conserved in Gross solution at a temperature of 4° C. does not give one hundred per cent results and is often complicated by thrombosis in the graft itself.

Alo-plastic material seems to be better, but we have not used it frequently enough to be able to give any opinion on it.

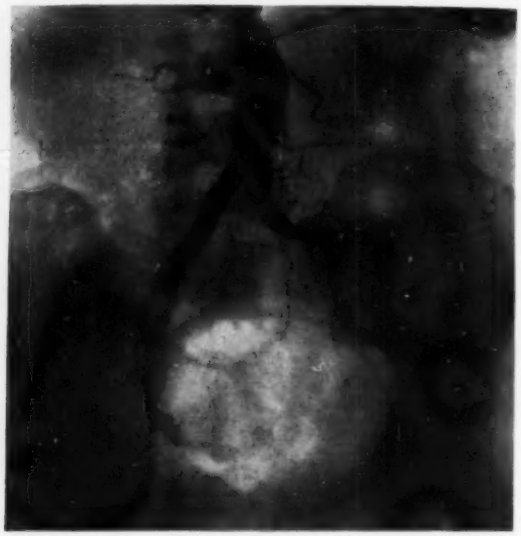


FIG. 699.—Post-operative aortograph (early phase) showing the proximal anastomosis. The calibre of the artery is quite satisfactory.

In the following case the arterial defect was overcome in an unusual way which proved to be successful.

CASE REPORT

The patient, M. M., a 48-year-old woman, was admitted to the Surgical Clinic on Sept. 18, 1953, and again on Oct. 10, with the diagnosis of aneurysm of the common iliac artery.

On the first admission she reported having had acute left otitis and pneumonia in childhood, and periostitis of the mandible in 1951.

The second examination revealed dyspnœa and pains in the left thigh.

FIG. 700.—Specimen of the resected aneurysm.

ON EXAMINATION.—Nothing abnormal was observed in the internal organs. In the left hypogastrium there was a pulsating tumour the size of an egg. It was well defined, had a smooth surface, and did not move on palpation. A systolic murmur was heard coinciding with the heart-beat, and a vibration was felt over the tumour. The pulsation of the femoral and posterior tibial artery was normal. Blood-pressure, 130/90; urine, no albumin, no sugar; sedimentation rate, 25;

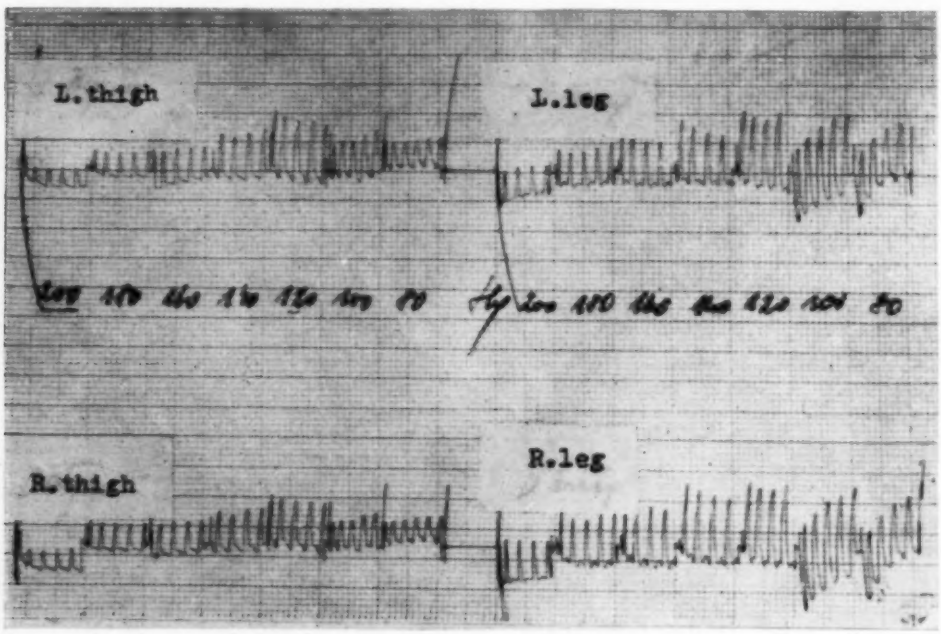


FIG. 701.—Oscillometric findings 3 years after operation.

red blood-cells, 4,300,000; hæmoglobin, 80 per cent. The white blood-picture showed eosinophilia and lymphopænia. W.R., negative. Chest radiographs showed an emphysema of the lung, a vertical position of the heart with a lightly dilatated aorta, and arteriosclerosis of the aorta. The E.C.G. examination showed nothing abnormal. The oscillometric findings on both thighs and legs were normal, and the skin temperature showed no marked difference between the right and left side. Aortography with 20 c.c. of 70 per cent. Joduron was performed through the translumbar percutaneous approach. It showed an aneurysm, the size of an egg, of the left common iliac artery (*Fig.* 697).

AT OPERATION (Oct. 21, under endotracheal anæsthesia).—The abdominal wall was opened through a pararectal incision from the edge of the last rib down to the inguinal region. The peritoneum was displaced medially. The left common iliac artery was found to be still normal ½ cm. below the aortic bifurcation; then it widened to a sausage-like shape 3 cm. in diameter and 6 cm. long. Below this point the artery was normal again and divided at a point 1 cm. distally into the external iliac artery and the hypogastric artery (*Fig* 698 A). The aneurysm was isolated and the whole common iliac, the external and the hypogastric arteries mobilized. Then resection of the aneurysm was performed (*Fig.* 698 B). Both ends of the iliac artery were closed with the U-suture. Afterwards the hypogastric artery was resected at a point of its peripheral bifurcation, the proximal end was brought upward, and the terminal lateral anastomosis between the hypogastric artery and the common iliac artery was performed (*Fig.* 698 C). The anastomosis functioned perfectly at the end of the operation. The retroperitoneal space was drained. Antibiotics were applied locally and the wound was closed in layers.

SPECIMEN.—This consisted of a 6 cm.-long sac with hard and friable walls. The intima was folded and easily removable from the media. The diameter of the sac amounted to 2 cm. (*Fig.* 700).

HISTOLOGY.—Panarteritis, probably of rheumatic origin. The media almost disappeared and the intima was thickened (productive endarteritis).

The post-operative course was uneventful. The patient was discharged 3 weeks after the operation with no disabilities.

On examination 3 years later (Oct. 9, 1956) it was found that the patient was leading a normal life. She walked normally; both extremities were warm, and pulsations of the peripheral arteries were normal. The wound was soundly healed; no murmur was heard. The oscillometric findings were normal (*Fig.* 701).

THE EFFECT OF HYPOPHYSECTOMY IN ADVANCED CARCINOMA OF THE BREAST

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DESPITE the value of radical surgery and radiotherapy there remain a large number of patients with advanced carcinoma of the breast in need of treatment. The endocrine palliation of breast cancer, and the concept of 'hormone dependent' tumours, are based on two premises (Huggins, 1956). The first is that the cancer cell may not be entirely independent of endocrine control; and the second that the normal endocrine environment is sufficient to encourage the growth of the cancer without the need of any abnormal hormone secretions. Many methods of changing the endocrine environment have been found effective in controlling breast and prostatic cancer, such as oöphorectomy, adrenalectomy, and the administration of œstrogens or androgens. Anterior pituitary hormones, in addition to those from the ovary and adrenal cortex, are known to be essential for physiological breast development. Presumably the most profound and prolonged alteration in body hormone pattern is the most likely to be therapeutically effective, and for this reason hypophysectomy has theoretical advantages (*Table I*). Luft, Olivecrona and Sjögren (1952) and Luft and Olivecrona (1953) first established hypophysectomy as a practical procedure, and their favourable observations were soon confirmed by Pearson, Ray, Harrold, West, Li, and Maclean (1954). Our report is based on observations up to April, 1957, following the earlier report by Baron (1956) concerning patients treated before November, 1955. In addition to the clinical aspects, which are given in detail, it is also necessary to discuss the problems of hormone dependency, and of completeness of hypophysectomy, tests of pituitary function, and the causes of the ultimate reactivity of the growth.

Table I.—CONTRAST BETWEEN THE ENDOCRINE EFFECTS OF HYPOPHYSECTOMY AND ADRENALECTOMY

Endocrine Activity	*After Adrenalectomy*	*After Hypophysectomy*
Growth hormone	Persists	Absent
Prolactin	Persists	Absent
Gonadotrophins	May rise	Absent
ACTH	May rise	Absent
Aldosterone secretion	Absent	Persists
Diabetes insipidus	Absent	Present
? Accessory pituitary	Persists	Persists
? Accessory adrenals	Persist	Atrophy

CLINICAL MATERIAL

We have studied the effect of pituitary ablation in 64 patients who have been observed for periods of up to 27 months. The conditions from which they were suffering are indicated in *Table II*. The effect of hypophysectomy in carcinoma of the prostate will

* Working under a full-time grant from the British Empire Cancer Campaign.

be described elsewhere. (Radley Smith, Gurling and Baron, 1958).

The majority were treated for carcinoma of the breast, and of these 52 have undergone surgical hypophysectomy and 2 pituitary stalk section alone. In collaboration with Sir Stanford Cade another 3 patients have had radioactive seeds inserted by the nasal route. Only the 52 cases subjected to surgical hypophysectomy will be discussed, including 1 patient

Table II.—Conditions for which Pituitary Ablation was Undertaken

	Cases
Carcinoma of the breast	57
Carcinoma of the prostate	4
Malignant melanoma	3
Total	64

who responded first to stalk section and later underwent hyophysectomy when her disease became active. All were suffering from advanced metastatic carcinoma of the breast, and many were in a deplorable condition at the time of operation, but as far as possible every patient thought capable of withstanding anæsthesia was accepted for treatment. The presence of pulmonary, liver, or bone-marrow metastases were not considered contra-indications. Their ages ranged from 24 to 69 years, with a mean of 53 years for the 52 patients under review, the age distribution being shown in *Table III*.

Table III.—Age Distribution in 52 Cases undergoing Surgical Hypophysectomy

Age	20–29	30–39	40–49	50–59	60–69	Total
No. of Cases	3	4	16	21	8	52

Previous treatment had included radical mastectomy in 39 cases, local mastectomy in 4, and radiotherapy in 44 patients. Five had previously undergone adrenalectomy, and 3 patients were referred because adrenalectomy was impracticable.

Grouped according to ovarian function, 17 (33 per cent) were premenopausal, 21 (40 per cent) were postmenopausal, and 14 (27 per cent) had undergone either surgical or irradiation castration. The response to operation will be analysed in relation to these findings.

PRE-OPERATIVE INVESTIGATIONS

As a rule each patient was admitted 3 to 4 weeks before operation to enable an accurate clinical assessment to be made and laboratory investigations to be undertaken. In some patients this pre-operative period was longer and devoted to securing improvement in their general condition, but 4 patients died whilst awaiting hypophysectomy.

The extensive radiological and biochemical investigations have previously been detailed by Baron (1956), and include liver function tests and serum and urinary calcium levels, in addition to those to be discussed later. Particular attention has been paid to the assessment of pituitary, thyroid, and adrenal function in order to correlate completeness or otherwise of hypophysectomy with clinical response, and to discern whether any recognizable change in hormone patterns was associated with regression of the tumour. The detection of adrenal insufficiency due to metastases also indicated the need for larger doses of cortisone at the time of operation. The tests upon which we assess pituitary function are summarized in *Table IV*. It soon became apparent that no single investigation could be relied on. The results of post-operative investigations will be reviewed later, and detailed reports of adrenal and thyroid function will be published elsewhere. Observations on the incidence of metastases in the anterior pituitary have been reported by Gurling, Scott and Baron (1957).

OPERATIVE TECHNIQUE

In most cases the pituitary is approached by the right frontal route, operating on the left side only when bone or cerebral metastases make it necessary. A truly frontal approach is much easier than that along the sphenoidal ridge, which is often used for pituitary tumours. On the other hand a severe degree of hyperostosis frontalis interna is common in women of this age, the inner table often being so adherent to the dura mater near the midline that it is torn in raising the skull flap, and is difficult to repair. The lateral route avoids this, but division of the pituitary stalk is more difficult. Using the frontal approach it is possible to elevate the frontal lobe to obtain a good view of the optic chiasma and pituitary stalk behind it. It is necessary to coagulate some of the superior cerebral veins running from the frontal pole to the superior longitudinal sinus, and if many of them are interrupted a dangerous degree of congestion may arise. Hæmorrhage into the frontal lobe, or extensive softening, may follow, or result from undetected cerebral metastases. Sometimes the anterior part of the right frontal lobe has been resected, giving a good exposure of the pituitary region. When the optic chiasma and nerves are first exposed quite large vessels may be seen anterior to the chiasma, but although they may be divided the optic pathways do not seem to be damaged. There is much variation in the length of the intracranial portion of the optic nerves and in the relation of the optic chiasma to the jugum sphenoidale, and when the chiasma is prefixed there may be insufficient room to remove the gland. After cutting the pituitary stalk and incising the diaphragma sellæ, the pituitary may occasionally be pulled out intact. Usually it is removed piecemeal using forceps, suction, and the appropriately curved Milligan tonsil dissector which enables the anterior portion to be scraped from under the clinoid processes. It may be necessary to remove a piece of bone between these two processes in some patients. We have not met severe bleeding from the inner wall of the cavernous sinus. As the gland is approached from above and in front it is the anterior part beneath the jugum which is most difficult to remove, and for this reason 5 unscreened radio-active gold seeds with a total activity of 15 m.c. are implanted with a Marsden-pattern gun. These ^{198}Au seeds (which emit β particles and γ rays) are kept in place with fibrin plaques above and below, deep in the fossa, 1·5 cm. below the optic pathways. The risk of visual damage seems less than with Zenker's fluid or after the less precise nasal implantation of radon or ^{198}Au seeds.

Anæsthesia has been a difficult problem because of the poor general condition of many of the patients, and because of lung metastases pleural or pericardial effusions, or irradiation fibrosis. The operation has been facilitated by hypothermia, which we have used for some time. External cooling is used after giving chlorpromazine and promezathine, nitrous oxide and oxygen, with a minimal amount of thiopentone during the operation. We consider it to be the best means of anæsthesia, and it has enabled severely ill patients to undergo hypophysectomy with greater safety.

Table IV.—Tests used in the Assessment of Pituitary Function before and after Hypophysectomy

Pituitary Hormones		Target Organ Function		General Effect
Gonadotrophins	Mammotrophic Hormone	Adrenal Function	Thyroid Function	Carbohydrate Metabolism
Urinary gonadotrophin excretion	Urinary mammotrophic potency (Hadfield test)	Urinary 17-oxo-steroid and 17-hydroxycorticoid excretion. Response to ACTH. Plasma 17-hydroxycorticoids. Post-operative cortisone withdrawal test. Blood eosinophils	Serum-cholesterol. B.M.R. ^{131}Iodine tests	Glucose tolerance. Insulin sensitivity. Glucagon sensitivity

PRE- AND POST-OPERATIVE CARE

Besides the usual pre-operative measures such as physiotherapy, blood transfusion, and aspiration of serous effusions, intramuscular cortisone is administered according to adrenal function, as indicated in *Table V*.

Table V.—Cortisone Dosage advised at the Time of Operation

Day	−2	−1	0	+1	+2	+3
Adrenal function	Dose of Cortisone (mg.)					
Normal	—	—	100 b.d.	50 b.d.	50 b.d.	25 b.d.
Impaired	100 b.d.	100 b.d.	200 b.d.	100 b.d.	50 b.d.	25 b.d.
Route of administration	I.M.	I.M.	I.M.	I.M.	orally	

On the second or third day it is usually given by mouth and continued indefinitely at 37·5–50 mg. daily in two doses. In patients who have previously undergone adrenalectomy we give 0·25–0·5 mg. of 9-α-fluorohydrocortisone by mouth daily for one week before and after operation, in addition to cortisone, obviating the need for extra salt and considerably simplifying electrolyte control. During the first 24 hours at least 1 litre of 0·9 per cent NaCl and 2 litres of 5 per cent glucose are given intravenously, with blood as required. In spite of diabetes insipidus with polyuria, fluid and electrolyte control is rarely difficult. In the event of severe hypotension, 100 mg. of hydrocortisone is given in 500 ml. of saline over a period of two hours, but if there is no response within 30–60 minutes, a minimal quantity of noradrenaline is added to the infusion. We give phenobarbitone 3 gr. (180 mg.) intramuscularly towards the end of operation, repeated 4 hours later to those patients operated upon under hypothermia, in whom very little thiopentone is used. Phenobarbitone ½ gr. (30 mg.) t.d.s. is continued for six months after operation. Symptoms of diabetes insipidus, which are present in under half the patients, are relieved by pitressin snuff, ¾–1½ gr. (15–30 units) as required, or pitressin tannate 0·5–1·0 ml. (2·5–5 units) by injection. Most need pitressin only at bed-time.

Oral cortisone is continued and increased to 25 mg. q.d.s. in the event of infection or accident, and the patient is warned of this before leaving hospital. Thyroid extract, 1½–3 gr. (90–180 mg.) daily, is given as soon as thyroid function tests have been concluded. The younger the patient the more thyroid appears necessary and the serum-cholesterol level is an aid to adequate dosage.

We have not found it necessary to give extra sodium chloride by mouth or to administer deoxycorticosterone acetate to patients with intact adrenals.

POST-OPERATIVE MORBIDITY AND MORTALITY

Recovery from the operation is usually as straightforward as after any craniotomy, and long-term complications have been few. Jacksonian and generalized fits can occur in the first 24 hours, but may not indicate serious brain damage, and in only 2 women have fits persisted after this period. Drowsiness lasting for 2 or 3 days may be ascribed to cerebral œdema in some cases, but it is not unusual for the patient to be out of bed after a week. Visual disturbances have been rare and in only two women has there been a permanent impairment of the visual fields, a bitemporal hemianopia in one and a quadrantic defect in the other. We attribute this low incidence in part to the use of ^{198}Au seeds rather than cauterization of the empty fossa with Zenker's fluid, as used by Atkins, Falconer, Hayward, and Maclean (1957), who observed field defects in 6 of their 30 cases. Loss of smell is a much more common disability of which we have become increasingly aware. It occurs in about three-quarters of our patients, recovers in a few weeks in some, and persists to a varying degree in one-half. The explanation is not clear since only the right olfactory nerve is divided, but it may be that traction on the contralateral olfactory nerve may damage its fibres. Loss of taste is rarely complained of, even in the presence of anosmia.

Immediate polyuria is apparent in 80 per cent of patients, and urinary output rises immediately after operation to 3–8 litres in 24 hours, followed sometimes by an 'interphase' between the fifth and tenth days in which the output falls toward normal (*Fig.* 702). Ray and Pearson (1956) have suggested that a clean cut of the pituitary stalk is less likely to be followed

by diabetes insipidus than diathermy coagulation, presumably because the cut end is able to release anti-diuretic hormone (A.D.H.). Eventually the stalk remnant appears to take over the function of the posterior lobe, polyuria gradually subsiding in all but a minority after six months to a year.

The 7 operative deaths were due to cerebral hæmorrhage in 4 instances, in one of which undiagnosed cerebral metastases were present, to extensive cerebral softening apparently due to venous thrombosis in 2 cases, and to pulmonary œdema in 1 other case. Two patients died within the first month, one of pulmonary insufficiency due to irradiation fibrosis, and another from increasing cachexia.

CLINICAL EFFECT OF HYPOPHYSECTOMY

To simplify the assessment of clinical response, numerical schemes, such as the 'mean clinical value' devised by Walpole and Patterson (1949), have been recommended by Atkins and his colleagues (1957).

Table VI.—SUMMARY OF RESULTS OF OBJECTIVE ASSESSMENT IN 52 WOMEN UNDERGOING HYPOPHYSECTOMY

	Number of cases	Percentage of total	Percentage of 33 surviving 3 months
Cases undergoing hypophysectomy	52	100	—
Survived three months	33	63	100
Improved { Regression	15	42	67
Improved { Arrest	7		
Unimproved	11	21	33

In our opinion these systems introduce their own fallacies and we have attempted to judge clinical response according to *subjective* and *objective* changes. Under *subjective* improvement we include relief of pain, increase in appetite, increased feeling of well-being, and, for example, reduction in dyspnœa without any clear-cut objective improvement. We have graded *objective* assessment as showing regression, arrest, or no change. By *regression* we mean definite measurable reduction in tumour masses, absorption of pleural effusions or ascites, diminution in size of the liver, or radiological recalcification of bone metastases. Under *arrest* we have classified those patients who have improved subjectively and undoubtedly had a worthwhile prolongation of life without significant change in physical signs or X-ray appearances. Since the previously extending growth is held in check, we consider it justifiable to group such cases together with those in whom regression has occurred, as *improved*. This relatively straightforward clinical evaluation is also used by Luft and Olivecrona and their colleagues (1956), and by Ray and Pearson (1956), and in our experience there is usually no doubt about the appropriate classification. Improvement is often apparent a day or two after operation, and the majority of patients who respond show signs of doing so within the first 2 or 3 weeks. Fever may abate and pain disappear immediately, troublesome effusions may begin to subside, and the patient is really given a new lease of life.

Since improvement may be transient in some patients and slow to become manifest in others, it seems wise to attempt an assessment only of those surviving for three months or more after operation. But it is essential to analyse the overall results in the 52 patients operated upon if we are to obtain, as it were, the bedside prognosis in the unselected case. Of the 52 patients undergoing hypophysectomy, 9 (17 per cent) died within the first month; in 7 death was associated with operation. There are 33 (63 per cent) who have survived for more than three months after operation in whom the effect may be fairly assessed.

There was objective evidence of improvement in 22 (66 per cent) of the 33 three-month survivors, in 15 of whom the growth regressed, and in 7 of whom it was arrested (*Table VI*). Subjective improvement was noted in 76 per cent of this group.

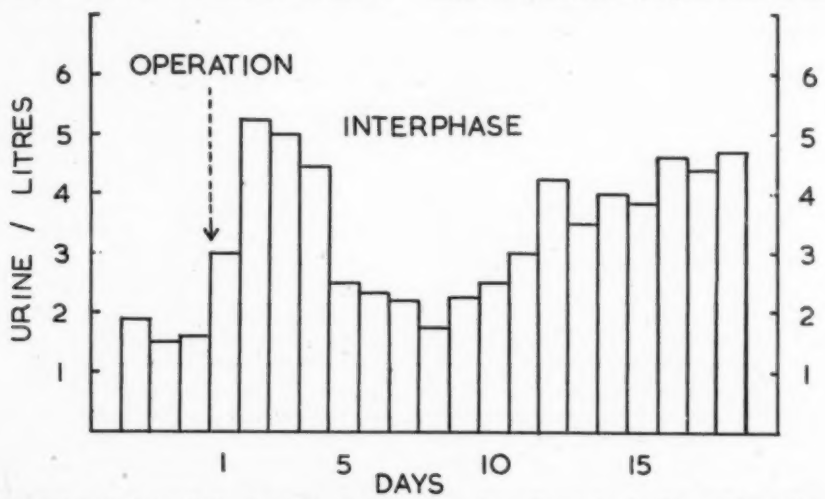

FIG. 702.—Urine volume before and after hypophysectomy, showing immediate polyuria, followed by an 'interphase' from the fifth to eleventh days when the output falls, and a return to high urine volumes.

Of the whole group of 52 there were therefore 42 per cent who showed objective evidence of improvement three months after operation.

Length of Survival and Duration of Remissions.—It is difficult to determine the expectation of life in the untreated patient, and a control series is lacking, but the length of survival of the failures who showed no improvement may be used as a

measure of prolongation of life in those who derived benefit from the operation. This, of course, assumes that those who failed to respond were in the same physical condition as those who did. The average length of survival after hypophysectomy in all 45 cases living one month or longer was 6·9 months (s.d. ± 6·4). For those showing no response survival averaged 3·3 months (s.d. ± 2·0), compared

Table VII.—Duration of Survival in 45 Patients surviving Operation for One Month or longer, according to Clinical Response

Effect of Hypophysectomy		Survival in Months					
		2–3	4–6	7–12	13–18	19–24	>24
Improved	Number	25	22	16	10	4	2
	Per cent	100	88	64	40	16	8
Unimproved	Number	20	11	3	—	—	—
	Per cent	100	55	15	—	—	—
Total	Number	45	33	19	10	4	2
	Per cent	100	73	42	22	9	4

with 9·8 months (s.d. ± 6·7) for those with objective improvement. It would appear that those showing improvement do in fact live longer than those not responding, and these figures will be exceeded since 12 patients are still alive and in remission.

$(t = 4{\cdot}6 \quad n = 45 \quad P<0{\cdot}01)$

The average duration of objective improvement was 8·6 months (s.d. ± 6·9) with a range of 1 to 24 months, that is, 1 month less than the average survival for the same group of patients. The terminal relapse often occurs with great rapidity. The duration of survival is indicated in *Table VII*.

Details of Clinical Response.—The most common problem was palliation either of the inoperable primary breast growth, often ulcerated, or of local recurrence in a mastectomy scar with lymph-node involvement. The frequency of some of these lesions, and the incidence of improvement, is shown in *Table VIII*.

Table VIII.—Incidence of Metastases and Post-operative Objective Improvement in 33 Patients surviving over Three Months

	Breast Primary or Recurrence	Bone Metastases	Lung or Pleural Metastases	Liver or Peritoneal Metastases
Number of patients	24	19	12	9
Number improved	14	6*	8	3
Per cent improved	58	32	67	33

* Excluding relief of pain alone.

Fourteen (58 per cent) of the 24 patients with breast involvement improved after operation, tumour masses and glands sometimes disappearing completely, whilst ulcerated lesions became covered with epithelium within a few weeks.

Mrs. V. P., aged 46 years, had been disabled by backache for three months before the diagnosis of mammary cancer was made. When admitted there was extensive ulceration of the primary breast growth and widespread bone and cutaneous metastases. Hypophysectomy relieved the bone pain immediately; the breast lesion became epithelialized by the eighth week (*Figs.* 703, 704); skin nodules on the leg regressed, and bone lesions recalcified radiologically (*Figs.* 705, 706). Her improvement has lasted over one year.

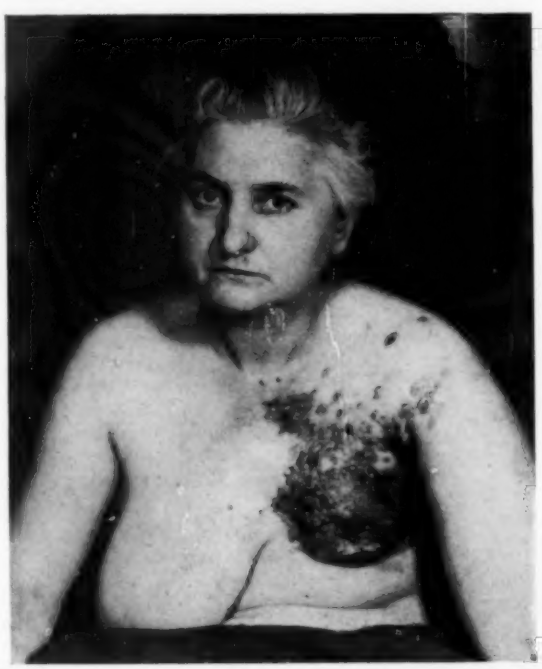

Fig. 703, 704.—Five months after hypophysectomy the extensive and ulcerated carcinoma of the left breast had become covered by skin epithelium and needed no dressings.

In some patients the breast lesion has healed satisfactorily whilst bone deposits have appeared unchanged in the radiographs. We have been

surprised by the speed with which pleural effusions have improved in some patients, and of the 12 with pleural or pulmonary metastases, 8 (66 per cent) have shown clear objective improvement. The X-ray appearances do not usually return to normal, but there is significant reduction in dyspnœa and the effusions have needed tapping infrequently or not at all. (*Figs.* 707, 708.)

FIG. 705, 706.—Two months after hypophysectomy there was radiological evidence of recalcification of metastatic deposits in many bones, including the left tibia.

Mrs. D. M., aged 32 years, had undergone radical mastectomy in 1953, and in 1956 became weak and increasingly dyspnœic owing to metastases with bilateral pleural effusions. She was extremely ill with widespread lymph-node involvement, deposits in the opposite breast and large pleural effusions needing aspiration every few days. Hypothermia anæsthesia was nearly abandoned because of her poor condition. She improved immediately after hypophysectomy, fever, tachycardia, and dyspnœa abating. Since then the effusions have not been tapped, the lymph-nodes and breast deposits have disappeared, and she has gained 40 lb. in weight. She is able to care for three children and undertake light part-time work some nine months after operation.

Bone metastases were known to be present in 19 of the 33 cases, but there was objective improvement, such as X-ray evidence of recalcification or reduction in size of a palpable lump, in only 6 (32 per cent). A higher proportion of those with pain experienced relief.

Mrs. H. W., aged 50 years, had bilateral local mastectomies for spheroidal-celled carcinoma in 1954, followed by radiotherapy. A year later she was found to have many bone metastases, including a large palpable deposit in the left occiput. In July, 1955, she underwent hypophysectomy with immediate relief of pain and regression of lymph-node metastases. The occipital metastases began to recalcify immediately. She remains well twenty-three months after operation.

The liver and peritoneum were involved in 9 of this group of three-month survivors, 3 of whom had ascites. Improvement was noted in 3 of them, and in 2 it was striking, the disappearance of ascites in one patient being slow.

Mrs. E. P., aged 48 years, had a radical mastectomy in 1948 for carcinoma of the left breast and remained well until 1954. She then developed a local recurrence, and metastases in lymph-nodes and liver. At the time of operation she was cachetic and jaundiced, with large hepatic deposits. Liver function tests showed serum-bilirubin 5·1 mg. per 100 ml., alkaline phosphatase 49 K.A. units, thymol turbidity 4·5 units, bromsulphthalein

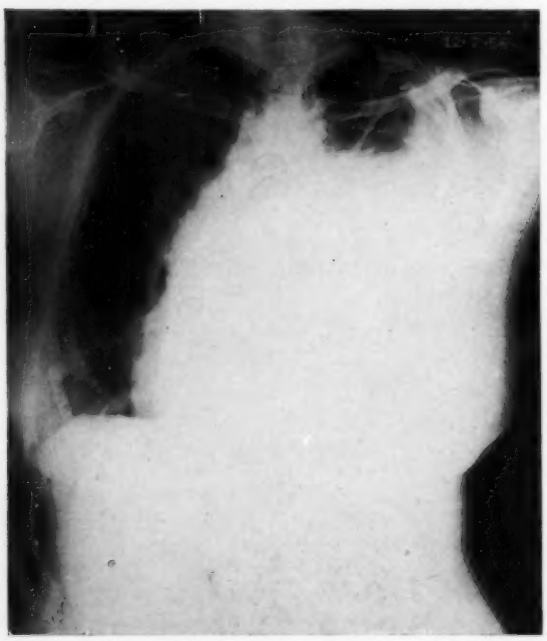

FIG. 707, 708.—Immediately after hypophysectomy there was rapid re-absorption of the pleural effusion as shown in the radiograph taken four weeks later.

retention at 45 minutes, 20 per cent. After hypophysectomy all evidence of metastases disappeared, liver function tests became normal, and she gained 56 lb. in weight. A year later she underwent appendicectomy for acute appendicitis, when the liver was found to contain numerous flat white fibrous scars. After 18 months she noticed a return of malaise, the liver enlarged, she became very jaundiced and died within a month.

FIG. 709, 710.—This emaciated patient, who also had diabetes mellitus, gained 56 lb. in weight six months after hypophysectomy. Regrowth of head hair is unaffected by the operation.

Disseminated skin lesions, pelvic masses, and dura mater metastases have also improved. The relief from pain, gain in weight, (*Figs.* 709, 710) and increase in appetite have often been striking. Malaise may disappear so quickly that it seems as if harmful humoral agents are immediately removed. There is no clinical differentiation to be made between these patients or in the lesions that respond favourably and those that do not. The failures, which are numerous and distressing, must not be forgotten.

Table IX.—CLINICAL RESPONSE ACCORDING TO AGE AT TIME OF OPERATION

	Age in Years					Total
	20–29	30–39	40–49	50–59	60–69	
Died in under 3 months	1	2	4	8	4	19
Improved	1	2	7	8	4	22
No response	1	—	5	5	—	11
Total	3	4	16	21	8	52

Influence of Age and Menstrual History.—As indicated in *Table III*, 8 (15 per cent) of our cases were over the age of 60 years, and 7 (13 per cent) were 39 years of age or less. In spite of the small numbers, it is of interest to determine whether age influences the incidence of response. According to Luft and Olivecrona and colleagues (1956), it is unlikely that patients over 60 years of age will improve, whereas Ray and Pearson (1956) consider their chance of responding to be good. In our experience there is no clear-cut difference in the proportion of successful cases at any age (*Table IX*).

It is possible that the age at operation does no more than reflect the menstrual status. When analysed according to menstrual history, 14 of the 33 three-month survivors were menstruating normally, 9 had passed the menopause, and 10 had previously had an artificial menopause. A higher proportion of premenopausal women improved after hypophysectomy than was the case in the postmenopausal group (*Table X*).

Table X.—RESPONSE IN 33 PATIENTS SURVIVING 3 MONTHS OR MORE, ACCORDING TO MENSTRUAL HISTORY

	Premenopausal	Artificial Menopause	Postmenopausal
Improved	12	3	7
Not improved	2	7	2
Total	14	10	9

These findings are at variance with those of Ray and Pearson (1956), who found a higher incidence of remissions in the postmenopausal group.

Response in relation to Previous Hormone Palliation.—Five patients had previously undergone bilateral adrenalectomy and oöphorectomy, two of whom had improved for 12 and 18 months respectively. In none of these patients was there a subsequent response to hypophysectomy (*Table XI*). Ray and Pearson (1956), who have hypophysectomized 11 adrenalectomized patients, thought that 4 derived

benefit from ablation of the pituitary. They concluded that it was a worthwhile procedure, particularly when the effect of adrenalectomy had been good.

Of the 12 patients who were known to have improved following castration or the administration of androgens or œstrogens, 8 (67 per cent) subsequently did well after hypophysectomy (*Table XII*).

Table XI.—Response to Hypophysectomy in relation to Previous Endocrine Surgery

Previous Treatment	Number of Patients	Improved after Adrenalectomy and/or Oöphorectomy	Responded to Hypophysectomy
Adrenalectomy and Oöphorectomy	5	2	0
Oöphorectomy	9	4	2
Total	14	6	2

On the other hand, there were 11 who had not improved, and of these only 1 (9 per cent) subsequently responded to hypophysectomy. The patient who benefits initially from hormone therapy will also improve after hypophysectomy if the tumour is still in the 'hormone-dependent' phase at the time of operation. When relapse occurs during or after hormone palliation or castration, the effect of hypophysectomy is less predictable.

We have found no connexion between the histological appearance of the growth and clinical response.

Post-operative Assessment of Pituitary Function.—In order to decide whether failure to respond or ultimate reactivity of the growth is due to the persistence or return of pituitary hormones, we have reviewed the results of tests of thyroid and adrenal function, urinary mammotrophic potency, and carbohydrate metabolism.

Thyroid Function.—The abolition of thyroid-stimulating hormone is followed by an immediate reduction in thyroid activity, and in most individuals clinical signs of myxœdema appear between the third and sixth weeks after operation, particularly in young patients. As two-thirds of our cases have shown signs of myxœdema before discharge from hospital, and since we have endeavoured to keep our patients as physiologically normal as possible, thyroid extract is given as soon as the investigations reveal evidence of impaired thyroid function. Atkins and others (1957) apparently did not find it necessary to give thyroid to all their patients. Adequate ^{131}I studies, including thyroid uptake, urinary excretion, and protein-bound iodine levels, were carried out on 40 patients. Evidence of myxœdema was obtained in 31 (77 per cent), and of a definite impairment of thyroid function in 6. In only 3 (7 per cent) did these tests reveal normal thyroid function. The basal metabolic rate was less decisive.

Although there was a fall in B.M.R. in 22 of the 33 patients investigated, in only 11 did it drop below the normal +5 per cent to −20 per cent range of the Aub-duBois method. The correlation between ^{131}I tests, B.M.R., serum-cholesterol levels, and clinical assessment of thyroid function is shown in *Table XIII*.

The serum-cholesterol was followed in all cases, the normal range being 140–260 mg./100 ml. In 24 (46 per cent) it exceeded this level and was a guide to the presence of myxœdema, but in 26 (50 per cent) the post-operative levels remained in the normal range. In another two the rise was very marked, yet did not go beyond the normal. Li and his colleagues (1955) considered the serum-cholesterol an unreliable indication of thyroid insufficiency immediately after hypophysectomy.

Table XII.—Previous Response to Endocrine Palliation in relation to Response to Hypophysectomy in Patients surviving 3 Months

Type of Palliation	Good Response to Previous Palliation	Good 3-mth. + Response to Hypophysectomy	No Response to Previous Palliation	No 3-mth. + Response to Hypophysectomy
Oöphorectomy	4	2	5	1
Œstrogens	2	1	3	0
Androgens	6	5	3	0
Total	12	8	11	1

Table XIII.—Comparison between Different Methods of Estimating Thyroid Function after Hypophysectomy

Thyroid Function	^{131}I Tests	B.M.R.	Serum-cholesterol	Clinical Assessment
Myxœdema	31	11	24	32
Reduced thyroid function present	6	11	2	9
Euthyroid	3	11	26	11
	40	33	52	52

There is reasonable correlation between thyroid function and other methods of determining whether pituitary destruction has been complete. The relation between post-operative thyroid function and clinical response is shown in *Table XIV*. Two patients had non-toxic thyroid enlargement before hypophysectomy and in both the goitre disappeared.

Adrenal Function.—Before operation adrenocortical function was found to be impaired, presumably because of metastases, in 3 patients besides the 5 already adrenalectomized. The increase in urinary 17-oxosteroids and 17-hydroxycorticoids after ACTH in the remainder was no different from that expected in patients with debilitating disease.

When 37·5 mg. or 50·0 mg. of cortisone is given daily, the levels of urinary 17-oxosteroids vary between 2·5 mg. and 8·5 mg. daily, even in the adrenalectomized patients, since 17–21–dihydroxy-corticoids are in part metabolized to 17–oxo-compounds. The value of some of the urinary steroid estimations are therefore limited and we have paid attention to the plasma 17-hydroxycorticoid levels during a 72-hour period of cortisone deprivation,

Table XIV.—Assessment of Thyroid Function after Hypophysectomy in relation to clinical Response in 33 Patients surviving over 3 Months

Assessment of Thyroid Function	Clinically Improved	Clinically No Response
Severely hypothyroid	20	8
Moderately hypothyroid	1	1
Euthyroid	1	2

measured by a modification of the procedure of Nelson and Samuels (1952) involving glucuronidase hydrolysis and separate estimation of free and conjugated fractions (Gurling, Gore, and Baron, 1958). Plasma levels fall more rapidly than urinary excretion, and the results appear to be more clear cut. When cortisone is withdrawn the symptoms and signs of adrenal insufficiency are not so obvious or dangerous as after adrenalectomy because aldosterone continues to be secreted.

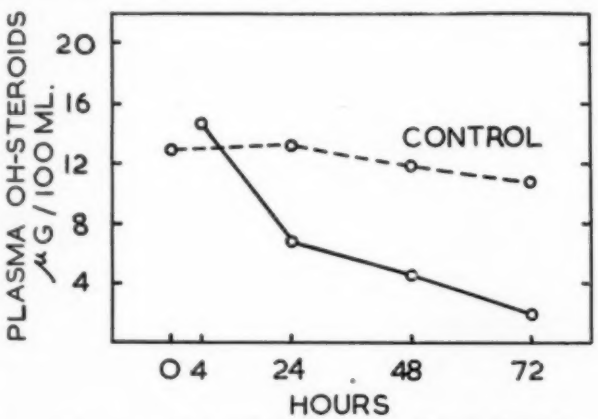

Fig. 711.—Cortisone-withdrawal test. If no cortisone is given there is a definite fall in the plasma-free 17-hydroxycorticoids after hypophysectomy. The control patient, although receiving a similar dose of cortisone for arthritis, showed no significant fall after omitting it for 72 hours.

Within 36–48 hours there may be anorexia, lack of strength, and sometimes mild postural hypotension with a slight fall in blood-pressure, tachycardia, and pyrexia. In the absence of ACTH secretion, urinary 17-oxosteroids and 17-hydroxycorticoids fall to less than 2 mg. a day. If hypophysectomy is functionally complete, the plasma-free 17-hydroxy-corticoids fall progressively when cortisone is withdrawn, to levels of 0–3 μg./100 ml. by 72 hours (normal range 8–18 μg./100 ml.) (*Fig.* 711) Baron (1958). Arthritic patients given similar doses of cortisone, in whom the pituitary-adrenal axis is intact, have in our experience had normal levels of free 17-hydroxycorticoids 72 hours after its withdrawal. Cortisone withdrawal tests have been carried out on 22 patients, and we have found that in 2 patients there was clinical improvement in spite of evidence of continued hydrocortisone secretion (*Table XV*).

Acute adrenal insufficiency has been observed in two patients taking 37·5–50 mg. of cortisone daily. On one occasion it was preceded by excessive sweating in hot weather, and in the other the patient had an attack of diverticulitis.

Table XV.—Post-operative Levels of Plasma 17-Hydroxycorticoids after Cortisone withdrawal for 72 hours, in relation to Clinical Response

Plasma 17-Hydroxycorticoids	Improved	No Response	Total
'Zero' levels	13	5	18
Normal levels	2	2	4

Differential 17-oxosteroid analyses have been performed on the urines of patients before and after hypophysectomy. Preliminary studies (Khattab, 1956) indicate that dehydro*epi*androsterone, which is secreted only by the adrenal cortex under the influence of ACTH, disappears from the urine after functionally complete hypophysectomy. These studies, and investigations of the relation of pre-operative 17-oxosteroid pattern to hormone dependence, as reported by Allen, Hayward, and Merivale (1957), are being continued with the collaboration of Dr. A. E. Kellie and Dr. A. P. Wade (Courtauld Institute).

Carbohydrate Metabolism.—We have not found either the glucose-tolerance or insulin-sensitivity tests to be of value in assessing completeness of hypophysectomy. The insulin sensitivity test carried out 4 to 5 weeks after operation is usually normal when cortisone is administered, and after omitting cortisone for 3 days definite hypoglycæmia has been found in only 3 patients, one of whom had liver metastases. Similarly the response to glucagon, with or without cortisone, has remained the same before and after hypophysectomy. It is apparent that the metabolic changes in the first few weeks after removing the pituitary are not comparable with those existing in well-established and untreated hypopituitarism.

Three of our patients had diabetes mellitus, one of whom had been taking 24 units of insulin for ten years, and the others mild diabetes of the lipoplethoric type not needing insulin. Carbohydrate tolerance improved in all. Insulin was discontinued in the first, the blood-sugar being normal when the daily dose of cortisone was 25 mg. or under; with doses of 75 mg., glycosuria reappeared and the blood-sugar rose.

Urinary Mammotrophic Potency.—A complex interaction of endocrine secretions is necessary for physiological breast development. Hadfield (1956) has claimed that the urine of premenopausal women contains a mammotrophic hormone, probably prolactin. He has devised a means of assaying mammotrophic potency in weanling male mice, in which the normally rudimentary breast is stimulated to an abnormal degree of duct growth and acinization. This test, he suggests, may be of value as a screening test for patients likely to respond to hypophysectomy. We have attempted to repeat his work, but in 25

women the response to hypophysectomy bore no relation to the presence or absence of urinary mammotrophic potency, using either unprocessed urine or a Klinefelter-type extract. The basis of this test seems insecure. Bahn and Bates (1956) have recently thrown doubt on the fact that prolactin is ever excreted in human urine, and it is difficult to see how the relatively large amount of prolactin necessary to produce breast growth in certain strains of mice could be present in as little as 2 ml. of human urine. It would, however, be premature at this stage to reject the validity of such a potentially valuable, though difficult test, and further studies are in progress.

Urinary Gonadotrophins.—Urinary gonadotrophin excretion has been studied in collaboration with Dr. Ian F. Somerville, Chelsea Hospital for Women, London, using the mouse uterus assay after kaolin extraction. Within the limits of the method the levels of urinary gonadotrophin showed a decline after operation in 29 (76 per cent) of the 38 patients investigated. When pre-operative levels were low, being 6 mouse units or less per day in 8 women pre-operatively, post-operative assays could be of no help in assessing pituitary function. On the other hand, levels of >384 m.u. have usually fallen to <6 m.u. by the third or fourth week.

Paradoxical results were obtained in 3 cases in which low initial levels increased after operation. We have not observed a resumption of high levels of gonadotrophin excretion at the time of relapse. We do not consider that gonadotrophin assay using the mouse uterus method is as accurate a means of detecting residual pituitary function as ^{131}I tests or plasma-steroid estimations. Two premenopausal women in whom urinary gonadotrophins were absent after hypophysectomy complained of hot flushes for up to three months after operation.

Other Investigations.—In many patients there was considerable improvement in hæmoglobin levels after operation, particularly when signs of leucoerythroblastic anæmia had been present. Blood regeneration did not appear to be deficient, and there was no clinical evidence in support of a pituitary hæmatinic factor. Serum-iron levels did not appear to change significantly. Plasma-protein levels sometimes became normal, and in particular a rise in albumin and fall in α_2-globulin was noted in paper electrophoretic studies. Hypophysectomy appeared to have no effect on the gastric secretion of hydrochloric acid.

Completeness of Hypophysectomy in relation to Clinical Response.—It is uncertain to what extent pituitary function must be impaired in order to achieve the metabolic changes responsible for regression of the carcinoma. If it could be shown that total hypophysectomy, in the absence of accessory pituitary tissue, was necessary for regression of the growth, whereas the presence of anterior lobe remnants was associated with lack of improvement, the interpretation of our results would be simple. Such is not the case. Sheehan and Summers (1949) have shown that in patients with classical evidence of hypopituitarism anterior lobe tissue is always present in the fossa. These anatomical pituitary remnants do not appear to possess significant biological function. Jacobsohn (1954) found that while pituitary transplants in the rabbit would survive when grafted beneath the temporal lobe, implantation in the midline and regrowth of hypothalamic vascular connexions were necessary before full endocrine function was restored. Are the anterior lobe remnants commonly found after hypophysectomy, of functional significance? Gomez and Turner (1937) claimed that as little as 2 per cent of residual pituitary tissue was sufficient to enable full breast development to be obtained in rats under certain conditions. Huggins (1956) states, "It is now appreciated that trace amounts of hormones can drive cancer to such exuberant growth that it causes death in the host". If only minimal quantities of pituitary hormones are necessary for continued division of the cancer cell, then the persistence of a few pituitary cells or a functioning pharyngeal pituitary (Boyd, 1956; Müller, 1957) might be the cause of failure to respond, and the regrowth of a small amount of anterior-lobe tissue might be responsible for relapse. Clinical observations do not support this conclusion. According to Forrest, Peebles-Brown, Morris, and Illingworth (1956), the implantation of radioactive seeds never destroys the anterior lobe entirely, but considerable clinical improvement can follow incomplete destruction. We have observed clear-cut regression of the cancer with 10 per cent and 25 per cent of anterior-lobe tissue in the fossa at autopsy. One case was considerably improved after operation limited to stalk section because of her poor general condition, but relapsed ten months later. An appreciable quantity of histologically normal pituitary tissue was removed at a second operation even though gonadotrophin and adrenal steroid studies did not then indicate pituitary function. She derived no benefit from the second operation, and at autopsy only a few pieces of fibrin debris remained in the fossa. Was reactivation of the tumour due to 'hormone independence' or to the persistence of the apparently functionless pituitary remnants? We must, however, agree that existing tests of pituitary function do not appear capable of detecting minimal activity.

It has also been shown that the peripheral production of certain hormones may be independent of pituitary or accepted target-organ function. Bulbrook and Greenwood (1957) and Greenwood and Bulbrook (1957) have confirmed that urinary œstrogens may be excreted after oöphorectomy, adrenalectomy and oöphorectomy, and after hypophysectomy, the completeness of which was not stated. The persistence of pituitary remnants with basal function may only be relevant to relapse, or failure to respond, if prolactin or growth hormone are essential for the multiplication of the cancer cell, a point as yet unproved.

POST-MORTEM STUDIES ON THE PITUITARY FOSSA

The pituitary fossa has been examined at autopsy in 17 patients, and an attempt has been made to make a rough estimate of the volume of anterior-lobe tissue remaining. Whole sections of the pituitary fossa after decalcification almost invariably reveal pituitary cells when the fossa appears macroscopically empty. These remnants are usually dispersed around the capsule, particularly under the jugum sphenoidale, suggesting that they derive their blood-supply from capsular vessels not arising from the superior or posterior hypophysial arteries.

Table XVI.—Results of Post-mortem Examination of the Pituitary Fossa with Estimation of Residual Anterior Lobe Tissue in relation to Endocrine Assessment and Clinical Response

A. Patients surviving over One month after Operation

No.	Patient	Age	Post-operative Survival (months)	Endocrine Function			Objective Clinical Response	Estimated per cent of Pituitary found at Post-mortem	Contents of Pituitary Fossa	Remarks
				Thyroid	Adrenal	Urinary Gonado-trophins				
1	G. S.	41	5	o	±	o	Arrest	<1	Empty naked eye. A few cells adherent to capsule	Screened ^{198}Au seeds
2	E. P.	48	18	o	o	o	Regression	<5	Small nodule of anterior-lobe tissue	Screened ^{198}Au seeds
3	D. E.	56	1½	±	o	o	No response	5–10	Scattered collections of tissue containing metastatic cancer cells	Screened ^{198}Au seeds
4	R. M.	28	27	±	+	+	Regression	25	Large piece of anterior-lobe tissue with remnant of pituitary stalk	—
5	M. H.	47	12	o	o	o	Regression	<5	Empty naked eye. Clumps of cells anteriorly attached to capsule	Screened ^{198}Au seeds Non-cancer death
6	A. C.	46	7	o	o	o	Short regression	<5	Empty naked eye. Scattered cells around capsule	Screened ^{198}Au seeds
7	R. V.	59	1½	o	o	o	No response	<5	Small nodule visible.	Screened ^{198}Au seeds
8	D. W.	52	2	o	o	o	No response	<5	Empty naked eye. Scattered subcapsular cells	Screened ^{198}Au seeds
9	F. H.	57	9	o	o	o	Regression after stalk section	20	0·1 g. of anterior pituitary was removed at re-operation	—
			10½	o	o	o	No response to hypophysectomy	<1	Empty naked eye; a few anterior lobe cells adherent to capsule	Unscreened ^{198}Au seeds
10	M. D.	54	5	o	±	+	Regression	<5	Empty naked eye. Thin layer of cells anteriorly attached to capsule	Screened ^{198}Au seeds
11	V. P.	47	12	o	o	o	Regression	<1	Empty naked eye	Screened ^{198}Au seeds Non-cancer death

B. Patients Dying within One month

12	D. S.	44	1 day	—	—	—	Operative death	−10	Small remnants visible naked eye. Early case; incomplete removal	—
13	D. D.	54	5 days	—	—	—	Operative death	5–10	Scattered nodules visible, histologically normal	Screened ^{198}Au seeds
14	F. S.	53	12 days	—	—	—	Post-operative death	5–10	One nodule visible. Scattered subcapsular remains	Screened ^{198}Au seeds
15	D. G.	45	13 days	—	—	—	Cancer death	<5	Empty naked eye. Scattered subcapsular cells	Unscreened ^{198}Au seeds
16	B. J.	62	17 days	—	—	—	Cancer death	5–10	Visible remnants. Small collections of cells anteriorly	Unscreened ^{198}Au seeds
17	F. B.	58	1 day	—	—	—	Operative death	<5	Empty naked eye. Few subcapsular clumps	Screened ^{198}Au seeds

As shown in *Table XVI*, patients dying within two weeks of operation tend to have more pituitary remnants than those who survive for longer periods. This may be due to the late effects of irradiation and impaired blood-supply, with progressive destruction of residual parenchyma. In all but two of the 11 women examined post mortem and who survived hypophysectomy for at least one month, there was no more than about 5 per cent of tissue remaining, and post-operative investigations failed to provide evidence of pituitary function. The small amount of tissue present months after operation tends to refute

the possibility of significant regeneration of pituitary tissue or reconstitution of the pituitary stalk and the hypophysial portal system.

There had been clinical improvement in 7 out of the 11, and no response in 2, one of whom had previously improved after stalk section.

Two patients were found to have about 10 per cent and 25 per cent of anterior pituitary tissue in the fossa post mortem. In one, Mrs. D. E., death occurred a month after operation without clinical improvement. Gonadotrophins had fallen from 24–96 m.u. to <6 m.u., and tests of thyroid function were equivocal. The second, Mrs. R. M., improved significantly after operation, but no radioactive seeds were inserted as she was one of our earlier cases. After operation thyroid and adrenal functions were present though depressed, and urinary gonadotrophin persisted at the 6–24 m.u. level 6, 18, and 24 months later. After 27 months she deteriorated rapidly and died. At autopsy the pituitary stalk was found to be present, together with the left anterior portion of the anterior lobe, which comprised about 25 per cent of the normal volume.

As already mentioned, Mrs. F. H. improved after stalk section, but relapsed; about 20 per cent of the anterior lobe had apparently survived and was removed at a second operation. We must conclude that clinical improvement can be achieved without total hypophysectomy.

Table XVII.—Comparative Summary of Response in Advanced Breast Carcinoma as shown by Published Series of Cases treated by Hypophysectomy or Adrenalectomy.

Hypophysectomy			Adrenalectomy		
Series	Number of Cases	Per cent Improved	Series	Number of Cases	Per cent Improved
Luft and others (1956)	52 (all cases)	42·3	Pyrah and Smiddy (1954)	22 (all cases)	45·4
	42 (selected)	53·6		21 (selected)	47·6
Ray and Pearson (1956)	74 (all cases)	48·6	Huggins and Dao (1955)	100 (all cases)	38
	67 (selected)	53·3		95 (selected)	40
Kennedy (1956)	28 (selected)	64	Cade (1955)	56 (all cases)	54·1
Present series	52 (all cases)	42·3		48 (selected)	46·4
	33 (selected)	66·6	Taylor (1956)	60 (all cases)	36
			Lipsett and others (1957)	70 (all cases)	51·4
				67 (selected)	53·7

METHODS OF PITUITARY DESTRUCTION

Surgical removal is at present the most effective means of pituitary destruction. The pituitary stalk is cut, the superior hypophysial arteries are interrupted, the bulk of the gland is removed, and radioactive seeds are inserted under direct vision. The acrylic plug recommended by Atkins and colleagues (1957), can also be put in place, but does not seem necessary in practice. Luft and Olivecrona (1955) and Ray and Pearson (1956) prefer to cauterize the fossa with Zenker's fluid or caustic soda. With these techniques it should be possible to leave no more than 1–5 per cent of histologically recognizable pituitary tissue in the fossa.

Pituitary stalk section alone has been advocated by Russell (1956). It seems probable that pituitary necrosis is in part due to infarction following interruption of the superior hypophysial vessels when the stalk is cut low in its course. In our 3 cases a relatively large amount of pituitary tissue appears to have survived. Campbell and Harris (1957) have found that in animals as much as one-third of the anterior lobe may survive. The operative mortality in stalk section and hypophysectomy appears to be the same. According to Luft, Olivecrona, Ikkos, Nilsson, and Ljunggren (1956) operative mortality after hypophysectomy was 9 per cent in their series of 52 cases, compared with 6 per cent of 74 cases reported by Ray and Pearson (1956). In our series deaths attributable to, or following operation, were 7, or 13 per cent. Atkins and others (1957) had no deaths in 30 cases.

The transnasal implantation of ^{198}Au or ^{90}Y seeds advocated by Forrest and his colleagues (1956), and by Greening (1956), may be less traumatic but the degree of pituitary necrosis is variable. Young (1957) has confirmed the limited effect of such seeds and calculated that a minimum of 70,000 r of γ-irradiation is necessary to destroy the parenchyma.

Lawrence and Tobias (1956) have induced pituitary destruction in animals and man using external irradiation with high-energy deuterons and report good results after treatment of 14 patients with breast cancer.

No adequate long-term comparison between these various methods of pituitary ablation has yet been reported.

CONCLUSION

We believe that hypophysectomy is probably the most effective means at present available of palliating advanced carcinoma of the breast.

Although it is not yet possible to decide whether the degree and duration of improvement is greater than after adrenalectomy, the post-operative management of the hypophysectomized patient is less difficult and stress is better withstood. Atkins and others (1957) attempted to decide which procedure is

the better by careful comparison of two similar groups, but their method of presenting results precludes comparison with other series. They concluded that hypophysectomy appeared to be more effective than adrenalectomy and oöphorectomy, but no statistical difference could be found. Luft and his colleagues (1956) recently reviewed the literature on hypophysectomy and collected reports on 197 cases, 110 of whom (56 per cent) were improved by operation. They considered that 53 per cent of their own 41 evaluated cases had improved. As shown in *Table XVII*, Ray and Pearson (1956), and Kennedy, French, and Peyton (1956) have reported much the same incidence of improvement. Our results are similar, 42 per cent of the whole group and 66 per cent of those surviving over three months having benefited. Figures concerning the length of remissions also agree. In a minority of patients the improvement lasts more than two years, with an average duration of about ten months, and exceptionally as long as three years. There is little reason to doubt that surgical removal of the tumour and radiotherapy remain the primary treatment of the early case of breast carcinoma. We suggest that although hypophysectomy is unlikely to become a routine operation it has a definite place in the treatment of the advanced case, and in addition may contribute towards the elucidation of cancer metabolism and the problem of hormone dependency. There seems to be no way of determining before operation which patient will respond favourably, and if such a test could be devised it would be of great help. But so-called 'hormone dependency' appears to be too complicated a problem to permit any one test, such as the mammotrophin assay described by Hadfield (1956), the urinary calcium excretion test of Emerson and Jessiman (1956), or the urinary differential 17-oxosteroid estimations suggested by Allen and others (1957), to give a definite answer.

Pituitary ablation should only be considered the ultimate treatment for advanced carcinoma of the breast if prolactin, somatotrophin, or other pituitary fractions could always be specifically implicated in the progression of the disease, or when it can be shown to be clinically superior to all other forms of therapy. We are not yet able to make this claim with certainty, and the search for other forms of therapy must continue.

SUMMARY

1. The effect of pituitary destruction has been studied in 64 patients with advanced malignant disease, 57 of whom had cancer of the breast. Surgical hypophysectomy was carried out on 52 of the cases, 2 had pituitary stalk section, and 3 radio-active seed implantation.

2. Surgical hypophysectomy with the insertion of 15 m.c. of unscreened ^{198}Au or ^{90}Y seeds is considered the best method of pituitary ablation.

3. Of the 52 patients 42 per cent showed objective evidence of improvement, or 66 per cent of the 33 who survived 3 months or more. The average length of remissions was 8·1 months, the longest being 27 months, and 12 patients are still alive. There appeared to be a prolongation of life from an average of 3·2 months for patients showing no response to an average of 10·1 months in those showing improvement.

4. Total ablation of all anterior pituitary tissue is rarely possible, but clinical improvement has been observed with between 10 and 20 per cent of tissue in the fossa. There is little evidence in favour of the regrowth of the pituitary stalk or of gland parenchyma.

5. The results of post-operative tests of pituitary function have been reviewed and correlated with post-mortem examination of the pituitary fossa.

6. Hypophysectomy is an effective method of palliating advanced metastatic cancer of the breast and prostate, and preferable to adrenalectomy, but it should not be considered a routine procedure. It may contribute materially to the elucidation of cancer metabolism and hormone dependence. The search for new methods of treatment must continue.

Note: Studies on 80 cases have confirmed the proportion of objective remissions, and suggest a greater length of remission than given above.

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HÆMOPERITONEUM

A RECORD OF 129 CONSECUTIVE PATIENTS WITH NOTES ON SOME UNUSUAL CASES

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SOME unusual examples of intraperitoneal bleeding which have been encountered recently have stimulated the authors to review all patients with this condition in the Oxford group of hospitals over the last six years (1951–1956), a total of 129 cases. We have excluded from consideration conditions merely producing blood-stained extravasations of fluid, (for example, acute pancreatitis and mesenteric thrombosis), and included only those patients where an extensive collection of frank blood was found either at operation or post-mortem. The aetiology and mortality of these cases, together with the accuracy of pre-operative or pre-autopsy diagnosis, is shown in *Table I*.

Of the patients in this series 87 (68 per cent) had bleeding of gynæcological origin (ruptured ectopic pregnancies or ruptured follicular cysts) and 30 cases (23 per cent) followed trauma to spleen, liver, or both these organs. There were also 4 ruptured aortic aneurysms leaking into the peritoneal cavity and 2 severe post-operative hæmorrhages. The 6 remaining examples, all of unusual origin, will be considered in detail later.

As regards accuracy of diagnosis, it was evident in nearly all cases that an intra-abdominal disaster had occurred. The exceptions occurred in the 8 patients dying after splenic and/or hepatic trauma. All these were admitted with overwhelming and rapidly fatal head injuries, the abdominal lesions being found incidentally at autopsy, often accompanied by multiple injuries elsewhere.

The common gynæcological emergencies and the traumatic hæmorrhages uncomplicated by head injuries were correctly diagnosed pre-operatively in the majority of cases (87 per cent), as can be seen from *Table I*. Of 74 patients where this information was available, the general practitioners had reached a correct diagnosis in 50 per cent of cases, and had invariably recognized the urgency of the condition with which they were called upon to deal.

The single fatality in the cases of gynæcological origin was unusual and of interest. She was a girl of 17 with known thrombocytopenic purpura who had had a splenectomy 5 years previously, but whose bleeding tendency had persisted. She died shortly after reaching hospital and autopsy revealed a massive hæmoperitoneum due to hæmorrhage from a ruptured follicular cyst.

There was only 1 death among the 22 patients with traumatic hæmoperitoneum uncomplicated by head injury. In this case the principal cause of hæmorrhage was a tear of the middle colic vessels, but, as well as this, there were lacerations of liver, colon, duodenum, and kidney.

All 4 patients with intraperitoneal rupture of aortic aneurysms were undiagnosed and all died. Of the 2 post-operative hæmorrhages, one followed a pancreatectomy for carcinoma and was fatal, the other followed appendicectomy and the patient recovered after re-exploration and blood transfusion.

The rare cases, not surprisingly, were all incorrectly diagnosed before surgery or autopsy and the mortality in this group was high, there being only 1 survivor in the 6 patients we now wish to consider in further detail.

Case 1.—**Spontaneous rupture of primary carcinoma of liver—laparotomy and packing of tumour—death.**

C. B., a male aged 68 years (R.I. 55059, P.M. 554/56), was admitted as an emergency under Mr. A. Elliott-Smith. He gave a history of 8 hours' severe abdominal pain which radiated into the back and which was accompanied by some vomiting. In recent months he had had mild indigestion and loss of weight.

On examination he was cold, sweating, and in great pain. T. 97°, P. 110, B.P. 140/90. The abdomen was rigid, uniformly tender, and silent. A diagnosis of

perforated peptic ulcer was made. However, laparotomy revealed more than 2 pints of blood in the peritoneal cavity and the right lobe of the liver was found to contain a large tumour which had ruptured through its diaphragmatic surface and which was bleeding freely; no obvious primary growth could be detected elsewhere. The tumour was packed with Oxycel gauze and covered with omentum. Three pints of blood were transfused, but the patient never rallied from the operation and died the following day.

Table I.—Final Diagnosis, Accuracy of Clinical Diagnosis and Mortality in 129 Consecutive Patients with Hæmoperitoneum 1951–6

Final Diagnosis	No. of Patients	Deaths	Correct Clinical Diagnosis
(*a*) *Gynæcological**—			
Ruptured ectopic pregnancies	77	0	70 (91 per cent)
Ruptured follicular cysts	10	1	4 (40 per cent)
(*b*) *Traumatic*—			
Ruptured spleens	21	4	17 (81 per cent)
Ruptured livers	6	2	4 (66 per cent)
Ruptured liver + spleen	2	2	0
Ruptured liver + middle colic artery	1	1	0
(*c*) *Other causes*—			
Intraperitoneal rupture of aortic aneurysm	4	4	0
Post operative	2	1	2
Unexplained at laparotomy	2	1	0
Ruptured primary tumour of liver, rupture of stomach in hæmatemesis, bleeding retroperitoneal varices, and hæmorrhage from juxtapancreatic vessel in hypertensive patient. (one example of each).	4	4	0
Total	129	20 (16 per cent)	97 (75 per cent)

* These exclude 36 unruptured ectopic pregnancies or clotted pelvic hæmatoceles and 15 ruptured follicular cysts with little or no bleeding.

Fig. 712.—*Case* 1. A section through the liver showing the hæmorrhagic necrotic growth on the upper surface. Many outlying nodules are seen, as well as portal vein branches plugged by tumour tissue.

At autopsy the liver tumour was found to be a necrotic and hæmorrhagic growth 7 cm. in diameter (*Fig.* 712) which had invaded the portal vein. No other tumour was found in the body in spite of a careful search.

Histological examination revealed an anaplastic carcinoma with marked pleomorphism and many giant cells (*Fig.* 713). No cirrhosis was found, but, nevertheless, this was considered to be a liver-cell carcinoma (hepatoma).

Comment.—Primary carcinoma of the liver is rare in white people. Stewart (1931) recorded an incidence of 0·1–0·3 per cent of all necropsies; 89 per cent of these were associated with cirrhosis of the liver. Schnabel (1935) encountered 1 case of hæmorrhage from a carcinoma of the liver in 27,000 autopsies.

Specter and Chodoff (1950) recorded a solitary case of hæmorrhage from a liver secondary. They collected 13 other examples of hæmorrhage from liver tumours in the literature up to that time, but it is particularly interesting that all apart from their own were primary liver tumours. The rarity of hæmorrhage from secondary hepatic deposits is unexplained.

Since 1950 three further reports have appeared (Mazingarbe, 1950; Riddell, 1952; and Roe, 1952) bringing the total number of examples of this condition we have been able to trace, including our own, to 18.

Case 2.—**Hæmatemesis from gastric ulcer—fatal rupture of the distended stomach.**

A. S., a female aged 77 years (R.I. 210647, P.M. 477/55), was admitted to the Radcliffe Infirmary under Mr. A. Elliot-Smith with a severe hæmatemesis, having already been given a transfusion of 10 pints of blood at another hospital without effect. On admission her condition was desperate. An intra-arterial transfusion was set up and nearly 6 pints of blood were given rapidly, but she failed to respond and died shortly after.

At autopsy there was a large hæmoperitoneum. The stomach, which had become distended with blood from

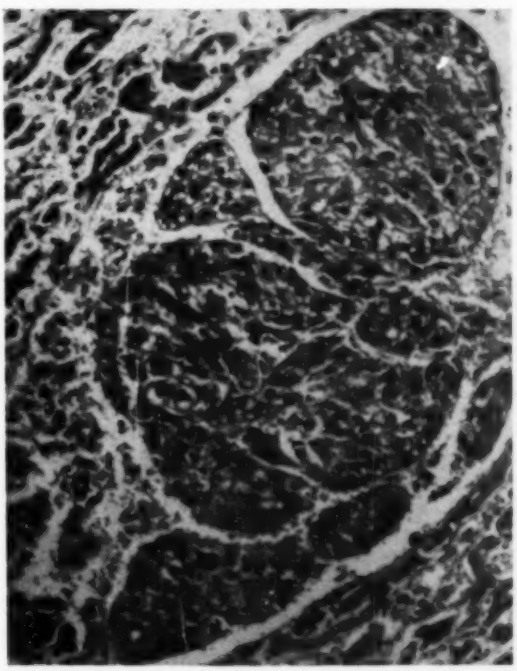

Fig. 713.—*Case* 1. A nodule of the hepatoma surrounded by compressed liver cells.

a vessel in the base of a chronic, posterior, benign gastric ulcer, had ruptured near the cardia, the tear being 3 in. long.

Comment.—Although it is not unusual to find at operation, or even on clinical examination, that the stomach is distended with blood and clot in a severe hæmatemesis, rupture of the stomach due to over-distension with blood appears to be exceedingly rare. Bolt and Hennessy (1955) report a case similar to our own, a female of 55, bleeding violently from a gastric ulcer, whose stomach ruptured with fatal consequences. They also record a second case in which post-mortem on a woman of 74, dying after a hæmatemesis, revealed several tears involving mucosa only, without, of course, a hæmoperitoneum. These authors could find no other examples in the literature, although spontaneous rupture of the stomach and tears due to instrumentation have been recorded.

As regards the aetiology of rupture of the stomach in hæmatemesis, Bolt and Hennessy consider that three probable factors are: violent and repeated vomiting efforts, contractions of the stomach in an attempt to expel clots, and rapid replacement of blood. It would seem advisable, therefore, to empty the stomach when there is clinical evidence of its distension in cases of hæmatemesis.

Case 3.—**Fatal hæmorrhage from retroperitoneal varices associated with cirrhotic liver.**

J. F., a retired policeman aged 74 years (R.I. 26056, P.M. 515/56), was admitted under Professor L. J. Witts with an 8-hour history of severe central abdominal pain and repeated vomiting. On examination he was profoundly shocked, with cold and clammy skin. The pulse rate was 100, the temperature subnormal, and the blood-pressure 60 mm. Hg. The abdomen was distended, with generalized tenderness but with only slight guarding; there was no clinical evidence of ascites. He failed to respond to resuscitation measures and died shortly after admission.

Autopsy revealed a massive hæmoperitoneum. The liver showed coarse cirrhosis and weighed 1975 g.; the spleen, weighing 355 g., had a greatly thickened capsule. The source of the bleeding was found to be a cluster of varices lying in front of the right kidney, the retroperitoneal hæmatoma thus formed having ruptured into the abdominal cavity. Numerous large œsophageal varices were present, but these had not bled. Microscopical examination confirmed the cirrhotic changes in the liver, which showed well-marked portal fibrosis and massive round-cell infiltration.

Comment.—We could find no comparable case in the literature. Palmer and Leitch (1945) have recorded a fatal intraperitoneal and intrapleural hæmorrhage from what were considered to be congenital varices on the upper and lower surfaces of the diaphragm. Their patient, a coloured male, aged 44 years, had a syphilitic aneurysm of the ascending aorta but no hepatic disease. James (1930) has reported a retroperitoneal hæmorrhage found at laparotomy on a patient with cirrhosis. There was no intraperitoneal bleeding in this case and recovery followed.

Varices, other than those in the œsophagus, are rarely a danger to the patient and, indeed, are beneficial in short-circuiting the portal obstruction. The case we record was a rare exception to the rule.

Cases 4 and 5.—**Two examples of 'spontaneous' intraperitoneal hæmorrhage.**

Case 4.—F. E., a lady aged 42 years (R.I. 1839), was admitted under Mr. A. Elliot-Smith with acute abdominal pain of several hours' duration. She was diagnosed as having a perforated peptic ulcer and submitted to laparotomy. This revealed an extensive hæmoperitoneum but a careful search failed to demonstrate the source of bleeding, all the intra-abdominal viscera being macroscopically normal. She was transfused and made a smooth recovery from operation. There was no clinical cardiovascular abnormality, her B.P. was 120/80, and, post-operatively, her bleeding time and platelet count were checked and found to be normal. She has remained well now for 4 years.

Case 5.—M. P., a labourer aged 35 years (R.I. 224517, P.M. 630/56), was admitted to hospital in a neighbouring town on June 17, 1956, having had a hæmatemesis severe enough to require transfusion. He gave a history of 12 months' indigestion but a barium meal had recently been carried out and showed no abnormality. Eight days after admission he developed a sudden severe abdominal pain, became shocked, and was thought to have perforated a peptic ulcer. At operation, however, he was found to have an extensive hæmoperitoneum for which no cause could be found.

Two further episodes of marked peritoneal irritation occurred 9 and 11 days after the operation and were treated conservatively; presumably these were further hæmorrhages.

At this stage he was transferred to the Radcliffe Infirmary under the care of Mr. D. C. Corry. On admission he was pale and thin, with a healthy, healing abdominal scar but no other clinical abnormality. His B.P. was 130/90. A complete hæmatological examination, including platelet-count and bleeding, clotting, and prothrombin times, was normal apart from showing a mild iron-deficiency anæmia. The stools showed large amounts of occult blood on repeated examinations.

On July 16, a month after his initial hæmatemesis, he developed acute abdominal pain, tenderness, and rigidity for the fourth time. The following day the abdomen was re-opened; again there was a hæmoperitoneum present but no cause for this could be found.

Splenectomy was performed, the spleen being macroscopically and histologically normal.

Following operation, he continued to have repeated attacks of severe abdominal pain. Repeated investigations were carried out, still without revealing any bleeding diathesis and, in spite of repeated transfusions, he slowly deteriorated, eventually dying on Sept. 11, nearly three months after his original admission to hospital.

Autopsy revealed a large left subdiaphragmatic collection of blood and there was also blood within the gastro-intestinal tract. The latter was easily enough explained because there were two shallow duodenal ulcers present. Careful search, however, failed to reveal the source of the intraperitoneal bleeding.

(Comment on these two patients follows the next case report which, itself, sheds some light on these examples of 'spontaneous hæmoperitoneum'.)

Case 6.—**Fatal 'spontaneous' intraperitoneal hæmorrhage. No apparent cause at autopsy. Fortuitous discovery of site of hæmorrhage on microscopic examination of vessels in relation to pancreas.**

J. Y., a chimney sweep, aged 58 years (R.I. 72307, P.M. 192/56), was admitted to the Wingfield-Morris Orthopædic hospital under Professor Trueta for surgical treatment of severe osteo-arthritis of the right hip. Apart from this local condition he was a healthy man, although clinical examination revealed a symptomless hypertension (B.P. 230/110). The cardiovascular system showed no other abnormality.

A pseudarthrosis of the right hip was carried out and all progressed well until the fourth post-operative day, when there was a sudden onset of severe central abdominal

pain accompanied by vomiting. Examination revealed a rapid weak pulse, and a generally tender, distended abdomen. The following day he appeared better, but 24 hours later there was a recurrence of pain and collapse and he passed into a state of irreversible shock and died.

At autopsy the site of operation on the hip contained a small hæmatoma but was not otherwise remarkable.

The peritoneal cavity contained a large blood-stained ascites, and also a large quantity of clotted blood. The source of this bleeding was a retroperitoneal hæmatoma near the head of the pancreas, where the clot was older and becoming discoloured. A careful anatomical dissection of the region showed no gross abnormality in any of the named branches of the cœliac axis or the superior mesenteric artery as far as they could be traced into the hæmatoma.

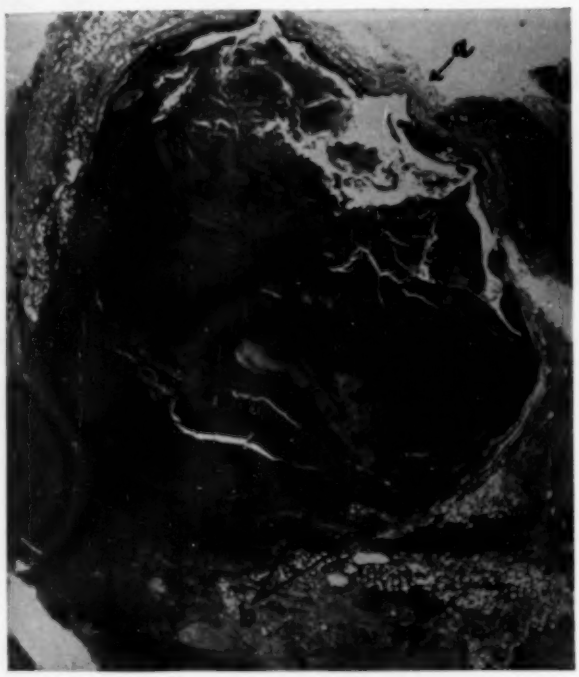

FIG. 714.—*Case* 6. The ruptured artery lying in the peripancreatic connective tissue. (*a*) Intact wall of artery; (*b*) Site of rupture of artery; (*c*) Hæmatoma in periarterial connective tissue.

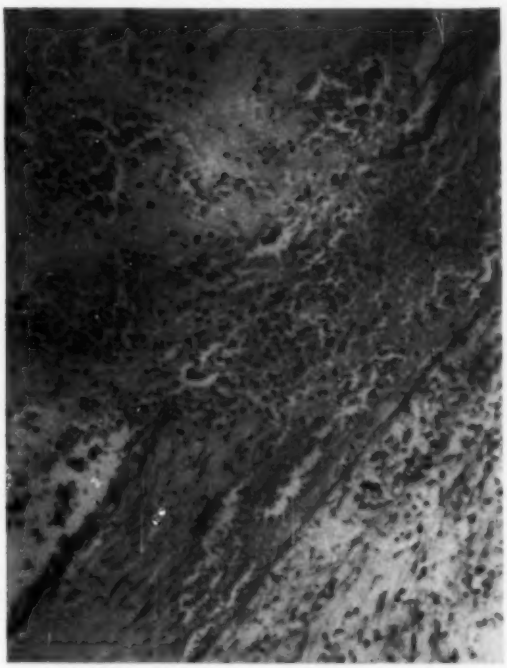

FIG. 715.—*Case* 6. A portion of the wall of the artery stained for elastic tissue. There is a large gap in the elastic lamina and polymorphonuclear leucocytes are infiltrating the wall.

Blocks of tissue were taken from the edge of the clot, at its apparent origin near the head of the pancreas, for microscopical examination and, quite by chance, one of these revealed the actual source of the hæmorrhage, a small artery which had obviously ruptured (*Fig.* 714).

The vessel showed marked aneurysmal dilation and its original diameter could only be surmised; it was perhaps 2–3 mm. Its wall was thinned and in places deficient, and appropriate stains showed patchy destruction of elastic tissue (*Fig.* 715). The lumen was obstructed by a thrombus which had burst through the defective wall and which showed early organization. The loose connective tissue around the vessel contained polymorphonuclear leucocytes, histiocytes and fibroblasts. There was some infiltration of the vessel wall with polymorphonuclear leucocytes.

The aorta, the branches of the cœliac axis, and the superior mesenteric artery did not show any histological abnormality.

Comment.—A number of examples have been reported in the literature of massive intraperitoneal hæmorrhage without obvious visceral disease or trauma. They are commonly referred to as examples of 'spontaneous hæmoperitoneum' or 'abdominal apoplexy', but it seems improbable that a vessel can rupture in the absence of an underlying organic lesion (Bunch and Madden, 1941).

In 1948, Brewer and Marcus reviewed the published cases of 'intra-abdominal apoplexy' and added an example of their own. They found that in 12 of the 28 patients studied, no definite bleeding point had been found; in the others several different vessels were implicated, including the right and left gastric, the superior mesenteric, gastroduodenal, gastro-epiploic, and middle colic arteries.

It is generally agreed that the commonest single cause of these so-called spontaneous hæmorrhages is arteriosclerosis, with or without hypertension. Brewer and Marcus found that 14 of their 28 patients were reported as arteriosclerotic, while Marks and Friedlander (1945) noted that 56 per cent of reported cases had hypertension or arteriosclerosis. The importance of these two conditions is also reflected in papers published more recently. Thus, Woolf and Thomson (1949) mention 2 patients with intraperitoneal hæmorrhage, both of whom showed marked hypertensive changes; Ross (1950) records the spontaneous rupture of a false aneurysm on the cæcal branch of the ileocolic artery in a hypertensive; and Sherwin and Gordimer (1950) describe hæmorrhage into the lesser sac from arteriosclerotic aneurysms of the splenic artery. In this connexion one notes that Sherlock and Learmonth (1942) found that 34 of 74 examples of splenic aneurysm showed arteriosclerosis and that 30 of the 56 cases of ruptured splenic aneurysm in the literature had burst into the peritoneal cavity.

Cases in which arteriosclerosis or hypertension were definitely absent are quite few in number. Brewer and Marcus (1948) found 8 in the literature (6 others were doubtful) and several have been reported since. Woodruff (1948) records a woman of 25 in whom an aneurysm on an omental vessel ruptured spontaneously; she was not hypertensive. Spontaneous hæmoperitoneum in an otherwise normal girl of 2 years is described by Jurishica and Vaccaro (1952); neither the source nor the cause was found. Kazmierski and Hellman (1954) report the spontaneous rupture of a lateral branch of what they term the 'colic cæcal' artery in a boy of 14.

The fact that these patients are often young adults or even children who are otherwise perfectly healthy is probably significant. It suggests that the hæmorrhage in some cases is the result of spontaneous rupture of a congenitally defective vessel wall, comparable with the well-known 'berry' aneurysms occurring on the cerebral vessels. That there is even some relationship between the two conditions is suggested in a paper by Nevin and Williams (1937), who describe 2 patients. In one a ruptured aneurysm on the splenic artery was associated with multiple aneurysms on branches of the circle of Willis; in the other a subarachnoid hæmorrhage from a single 'berry' aneurysm on the circle of Willis was associated with a massive hæmoperitoneum of uncertain origin.

These two conditions, arteriosclerosis and congenital vascular defects, probably explain most cases of abdominal apoplexy where this is not due to some more obvious cause. However, in patients with no evidence of arteriosclerosis, the diagnosis of some congenital vascular defect is often made on negative grounds. This latter situation is well shown by *Case* 5 in the present series. The patient was a young man and was not hypertensive. At autopsy no signs of arteriosclerosis were found. There was no gross evidence of any congenital vascular abnormalities, nor were any revealed by histology. Both the source and the cause of the hæmorrhage remain a mystery. The same is true of *Case* 4. This lady was also normotensive and at laparotomy no gross arteriosclerosis was seen. There is no evidence that she had any congenital vascular defects; no source was found for the hæmorrhage and she has since remained well.

Our last patient (*Case* 6) is an example of the group in which hypertension is the apparent cause, although in this man arteriosclerosis was not very severe. In this case sections of the pancreas showed in one place not directly related to the hæmorrhage, a small collection of inflammatory cells, and the possibility that a small localized area of acute pancreatitis weakened the vessel wall cannot be completely excluded. In the same way the bleeding might have been consequent on a mycotic aneurysm, but no likely source of an infected embolus was found.

The most interesting, and to us instructive, feature of this case was the fortuitous finding of the actual site of the hæmorrhage. In papers published in the past, some distinction has been made between those cases in which a definite bleeding point was discovered and those in which none was found. This distinction is probably virtually meaningless. *Case* 6 might easily have been put, with *Cases* 4 and 5, into the group with "no definite bleeding point", for a careful anatomical dissection had failed to find the source of bleeding. However, a block of tissue, taken for routine histological examination, happened to contain the affected artery. We suggest that the failure to find the bleeding point in some examples of 'abdominal apoplexy', both in the present series and in the literature, was not because some local focus did not exist, but because less good fortune attended the pathologist's investigation of these cases.

SUMMARY

1. The aetiology of hæmoperitoneum in 129 consecutive patients is analysed and the accuracy of diagnosis considered.

2. The great majority were gynæcological (68 per cent) or traumatic (23 per cent) in origin.

3. Examples of a number of rare causes are presented and discussed.

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EMERGENCY RESECTION AND ANASTOMOSIS FOR PERFORATED SIGMOID DIVERTICULITIS

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INTRODUCTION

RESECTION is the only certain cure for diverticulitis once complicated by abscess, fistula, obstruction, or free perforation. At the time of one of these complications most surgeons will perform a colostomy and excise the bowel later; Paul-Mikulicz resection is an alternative. Lepère (1955) alone points out that where immediate resection is necessary (for "gangrenous sigmoiditis") and the ends cannot be brought out, immediate anastomosis must logically be performed.

Impressed with: (1) The safety of resection and anastomosis in several personal cases of perforated colon carcinoma; (2) The fact that colostomy for perforating diverticulitis makes two more operations necessary (resection and closure of the colostomy)—often in elderly people; (3) The observation that later colectomy is sometimes made more, rather than less, difficult by mature adhesions and persistent inflammation (Lockhart-Mummery, 1938, and compare *Case* 6 of the present paper); (4) The fact that perforating diverticulitis is often quite localized and the sigmoid flexure mobile; the present author performed emergency resection and anastomosis in 4 cases of perforated sigmoid diverticulitis, selected from 9 seen in the same 2-year period.

These 4 patients are well and it is suggested that this radical treatment, where suitable, may save life and cure the disease in one stage.

HISTORY

Cruveilhier (1849) foresaw perforation: "On conçoit que ces petites cellules muqueuses puissent être irritées par la présence des matières fécales et devenir le siège d'inflammation et de perforation." Fiedler saw a perforation at post-mortem in 1868 and Regnier (1898) described the first case, a patient of Jaboulay's.

All 28 cases reviewed by Patel in 1911 died. By 1940, when colostomy with or without closure of the perforation and drainage was usual, the mortality was about 60 per cent (Arnheim, 1940; compare Eggers, 1931; Lazarus, 1933; Curran and Goodale, 1937). Antibacterial drugs have since reduced the number of deaths from peritonitis, but I suspect the overall mortality is still higher than that for perforated peptic ulcer. No considerable series has been described recently to my knowledge.

Resection for chronic sigmoid diverticulitis was first suggested by Mayo in 1907 (Young and Young, 1944). Treatment "is comprised in a single word—surgery" said Telling and Gruner in 1916, referring to two-stage removal of the bowel. Unfortunately, too many of the patients died, and surgery fell out of favour. Even in Lockhart-Mummery's series (1938) the mortality was 23 per cent and in Arnheim's (1940), 21 per cent. But Lockhart-Mummery's paper was of immense importance. It showed that the survivors of resection were cured. Shortly afterwards Smithwick (1942) demonstrated that those patients not resected were not cured; late follow-up after conservative management showed that 47·5 per cent were unwell and 12·5 per cent had died of the disease. When the mortality fell steeply following the use of antibacterial drugs (2·7 per cent, Pemberton, Black, and Maino, 1947; 2·6 per cent, Welch, Allen, and Donaldson, 1953) resection was properly confirmed as a safe cure for diverticulitis.

Though a colostomy used to be made as a preliminary, one-stage resection and anastomosis is now thought quite safe in the absence of acute complications (Moore and Kirksey, 1954; Provenzale, 1955; Colcock, 1956; Welch and Rodkey, 1956).

THE PATHOLOGICAL GROUNDS OF TREATMENT

Diverticula develop gradually and inexorably with age, so that two-thirds of those patients over the age of 85 have them (Welch and others, 1953). Fifteen per cent of these people will suffer diverticulitis (Pemberton, Black, and Maino). At any rate diverticula, once formed, persist; no conservative measure will remove them.

The appendix can empty itself and becomes inflamed only if obstructed. A diverticulum cannot empty itself; stagnation and inflammation can occur in it without obstruction. Thus an inflamed diverticulum may perforate while still communicating freely with the bowel which can still empty fæces into it and out of the perforation. Colostomy will control this flow of fæcal material and save life.

On the other hand colostomy, though it prevents further filling of a diverticulum, does nothing to empty it (Lloyd-Davies, 1953). Diverticulitis will persist, and perhaps grow worse, in spite of it. Colostomy is no cure. Rarely, a single diverticulum is inflamed (Spriggs and Marxer, 1925). Usually several are inflamed and one ruptures. More important, the almost invariable peridiverticulitis obscures the diverticula. It does not bury them—in fact each sac is near the surface (Telling and Gruner) and may perforate free into the general peritoneal cavity (*see Fig.* 718). Perforation is perhaps more likely in these cases; emptying is made almost impossible by elongation and distortion of each diverticular neck in thickened subserous fat (Telling and Gruner). However, such a diverticulum may rupture first into the enveloping mesocolon and produce a little abscess which points through the mesocolon (Lazarus). To 'close' this secondary perforation is to close an abscess, and is surgically unsound. At the same time anaerobic cellulitis of the mesocolic fat may take place; this is perhaps Lepère's "gangrenous sigmoiditis" (*see Fig.* 720). A colostomy, or 'closure of the perforation', would hardly affect the course of such an inflammation, once set up.

Too often, then, local treatment of perforated diverticulitis (excision or closure) has no sound basis

and is unsafe. More than one diverticulum is inflamed; the diverticula cannot be clearly defined; the visible 'perforation' may be merely a pointing mesocolic abscess; 'gangrenous sigmoiditis' defies conservative local measures.

THE INCIDENCE OF PERFORATION

Approximately a quarter of all cases of diverticulitis suffer surgical complications (Pemberton, and others, 1947). The figures for free perforation vary widely—from 1·8 per cent of all cases of diverticulitis in Telling and Gruner's series to 12 per cent of the 834 cases reviewed by Arnheim in 1940. Darling (1934), in Australia, described it as the most common complication, while it is not even mentioned by Welch and Rodkey.

MATERIAL

From December, 1954 to December, 1956, the author has treated 9 cases of perforated sigmoid diverticulitis with general peritonitis. All were primarily free perforations except one in which an abscess had secondarily ruptured into the general peritoneal cavity. All were operated upon. In two very ill patients drainage alone was carried out; both died. In one successful case the perforation was closed and the peritoneal cavity drained. Two patients recovered after drainage and transverse colostomy. The remaining 4 cases were successfully treated by immediate sigmoid resection and anastomosis.

CASE REPORTS

Case 1.—Mrs. S. L. H., aged 73, developed spreading peritonitis while in hospital with incomplete bowel obstruction due to a known diverticulitis. She had had mild obstructive attacks for three years.

At laparotomy on Dec. 22, 1954, I found a widely-adherent sigmoid inflammatory mass with free pus in the lower abdomen and pelvis. Transverse colostomy and peritoneal drainage were performed. She recovered quickly and remained well.

A year later the colostomy was closed (Mr. Leslie), but nine months afterwards severe pains recurred and the affected bowel was removed in two stages. She is now (Feb. 6, 1957) symptom-free.

FIG. 716.—*Case* 3. Longitudinal section of specimen showing the progressive diminution in size of diverticula towards each end.

Case 2.—Mrs. K. P., aged 68, had her first attack of pain in the right lower abdomen four months before admission. She had no bowel symptoms then or now. One day after the second attack began her temperature was 100° F., her pulse-rate 120; there was local tenderness and guarding about McBurney's point.

At operation on April 16, 1955, I sutured an appendix epiploica over a perforation in the lower sigmoid colon. There was some coliform pus in the pelvis, and a drain was inserted there. Recovery was straightforward, but we did not see her again.

Case 3.—Mrs. S. C. S., aged 76, had recurrent bowel colic with occasional diarrhœa and bright bleeding per rectum for one year. She took cascara twelve hours before admission and immediately felt severe constant pain all over the abdomen. Vomiting was profuse. Her whole abdomen was rigid and tender, her pulse-rate 100, and her temperature 99° F.

Operation on Nov. 10, 1955, revealed established general peritonitis due to perforation of a large sigmoid diverticulum. I resected the diverticulous colon, 6 in. in all (*Fig.* 716), and performed end-to-end anastomosis without drainage.

Recovery was uneventful. Follow-up barium enema on Feb. 6, 1956, showed 'no lesion in the colon' (*Fig.* 717). She is now (Feb. 6, 1957) symptom-free except for occasional slight 'wind pains'.

Case 4.—Mrs. D. E., aged 56, was admitted in diabetic pre-coma (blood-sugar 1081 mg. per cent) and with general peritonitis so advanced that the abdomen was distended and showed shifting dullness. She had had pains in the left lower abdomen on and off for three months, and severe constant generalized pain with frequent vomiting for seven days.

Huge doses of insulin failed to control her diabetes. At laparotomy on Jan. 3, 1956, I found advanced general peritonitis due to perforation of a sigmoid diverticulum and merely drained the pelvis. She died 32 hours after admission.

FIG. 717.—*Case* 3. Follow-up barium enema.

Case 5.—Mr. J. K., aged 72, was admitted after three months of intermittent bowel colic, with frequent vomiting and severe constant lower abdominal pain of 48-hours' duration. His abdomen was generally tender and rigid, his pulse-rate 120, and his temperature 101° F.

At laparotomy on Jan. 5, 1956, I found a large perforated sigmoid diverticulum and foul turbid fluid free in the lower abdomen and pelvis. The sigmoid loop

(8 in.) was resected (*Fig.* 718) and end-to-end anastomosis performed. Recovery was uneventful except for a wound abscess. Barium enema on March 6, 1956 (*Fig.* 719), showed only 'one or two isolated diverticula'. He is now (Feb. 6, 1957) symptom-free.

Case 6.—Mr. E. S., aged 44, had no symptoms until colic and vomiting began ten days before admission. Though he passed a little fæces each day, his abdomen

FIG. 718.—*Case* 5. Transverse section of specimen showing the diverticulum which had perforated. Though enveloped in thickened fat, its surface is subserosal (Telling and Gruner, 1916).

began to swell. An enema on the morning of admission caused sudden severe pain in the left iliac fossa. His temperature was 100·6° F., and his pulse-rate 102. His abdomen was distended, rigid and silent.

At operation on Jan. 26, 1956, I found fæces and pus about a lower sigmoid perforation and foul turbid fluid all over the abdomen; the small bowel, but not the colon, was distended and paralysed. Transverse colostomy and pelvic drainage were performed.

FIG. 720.—*Case* 7. Specimen showing 'gangrenous sigmoiditis' and secondary perforation of a mesocolic abscess.

A fæcal fistula at the drain site and persistent ill-health led to re-exploration by Mr. d'Offay on May 29, 1956, when a mass of fistulous ileum and sigmoid (containing *one* diverticulum only) was removed. Later his colostomies were closed and he is now (Feb. 6, 1957) perfectly well except for occasional 'wind pains'. He has had two attacks of diarrhœa at home.

Case 7.—Mr. C. B., aged 50, said he had had a suprapubic ache and diarrhœa for five years, but colic for the first time during the last week. Severe abdominal pain had begun that afternoon. He did not vomit and his bowels were loose. His temperature that evening was 100° F., pulse-rate 120; his abdomen was generally rigid and tender.

At laparotomy on Feb. 4, 1956, general peritonitis was found, due to perforation of a sigmoid diverticulitis via the mesocolon. There was foul purulent fluid throughout the peritoneal cavity. I resected a 5-in. segment (*Fig.* 720) of sigmoid colon and performed end-to-end anastomosis, draining the pelvis. He recovered quickly, but had an incisional hernia following a wound abscess. He is now (Feb. 6, 1957) quite well

FIG. 719.—*Case* 5. Follow-up barium enema.

FIG. 721.—*Case* 7. Follow-up barium enema.

but for occasional 'heaviness' on his right side. Barium enema on April 1, 1956 (*Fig.* 721), showed 'prediverticulosis' of the descending colon.

Case 8.—Mr. S. G., a frail man of 43, had been bedridden with ankylosing spondylitis for several years. He had had bowel colic on and off for two years. Thirty-six hours before admission he developed severe constant abdominal pain, with diarrhœa. His abdomen was generally rigid, but tender only in the right iliac fossa. His temperature was 99° F., and his pulse-rate 94.

At laparotomy on March 11, 1956, I found a perforated lower sigmoid diverticulum and purulent non-foul fluid free in the lower abdomen. The pelvis was drained but ileus persisted and he died on March 16, 1956.

Case 9.—Mrs. A. J., aged 59, had suffered from constipation and a suprapubic ache for many years. On the morning of admission she suddenly felt severe pain all over the abdomen and vomited several times. Her temperature was 100° F. and her pulse-rate 120. Her abdomen was board-like and most tender below the umbilicus.

FIG. 722.—*Case* 9. Specimen showing the free communication of perforated diverticulum with lumen of bowel.

At operation on June 24, 1956, I found a sigmoid diverticulitis perforated via the mesocolon, resected 7 in. of sigmoid colon (*Fig.* 722) and performed end-to-end anastomosis. Fæces and foul fluid had been free in the abdomen. Recovery was straightforward but for a wound abscess.

Now (Feb. 6, 1957) she feels well and is much less constipated than before operation. She had an attack of colic in August, 1956, after eating leeks, and another in January, 1957, after eating plums. Barium enema on Aug. 7, 1956 (*Fig.* 723), showed a persistent "local segment of diverticulitis in the upper pelvic colon".

NOTEWORTHY CLINICAL FEATURES OF THESE CASES

In one patient perforation followed a purgative, in another an enema (*see* Lazarus, 1933).

Four patients had an initial colic for hours or days. All 9 later suffered the severe constant pain of peritonitis, usually of sudden onset, but less often localized than in appendicitis. Only 2 complained of pain in the left and one of pain in the right lower abdomen. In the others pain was felt right across the lower abdomen, or was generalized.

Two patients were constipated (*Case* 1, of course, had intestinal obstruction as well); 2 had diarrhœa; 5 had no previous or present bowel symptoms.

Repeated vomiting was the rule, and is merely a characteristic of peritonitis—though uncommon, it should be remembered, with perforated peptic ulcer.

Eight patients had previous attacks of pain. Four had previous bowel irregularity. Not so much the length of history as its relative unimportance to the patient served to rule out a diagnosis of perforated carcinoma. In some cases the earlier symptoms were only admitted at later direct questioning.

It is interesting to analyse the pathological basis of symptoms in these 9 cases, and compare them with carcinoma. In carcinoma pain (colic) is due chiefly to narrowing of the lumen, marches with bowel irregularity, and is uncommon without it.

In diverticulitis, pain is due to distension (colicky pain) or inflammation (constant pain) of the diverticula themselves. Four patients had previous pains without bowel symptoms. In each of these cases there were one or few inflamed diverticula and little peridiverticulitis. Three patients had colicky pain, in the others the pain was constant. In the only patient (*Case* 6) with no previous symptoms at all, only one diverticulum was found in the subsequently resected sigmoid.

FIG. 723.—*Case* 9. Follow-up barium enema.

Those 4 patients with previous bowel symptoms all had more than one inflamed diverticulum. Those two with the longest histories each had 'secondary' perforation of a mesocolic abscess, indicating gross thickening due to peridiverticulitis.

If any conclusion is to be drawn from these observations, it is that those patients with bowel symptoms, particularly of long duration, are those with the most advanced diverticulitis and peridiverticulitis, and may have a type of perforation which cannot be dealt with by local measures. Resection is indicated on both counts. On the other hand, in those patients without bowel symptoms and presumably with one or few diverticula and little peridiverticulitis, closure of the perforation may be possible: whether it is a cure is open to question.

Only in the patient (*Case* 1) who was already known to have one, was a mass felt in the abdomen. In only one patient was a mass—tender of course—felt per rectum, and only two of the others had definite rectal tenderness, which is surprising.

Three patients had distended abdomens, one because of obstruction, two with severe advanced peritonitis. In the others the signs were those of an early general or spreading peritonitis—widespread rigidity and tenderness. In only one patient was tenderness sharply localized, and then at McBurney's point.

There seem to be two fairly distinct syndromes of perforated sigmoid diverticulitis—one in which there

are one or a few large diverticula, characterized by a short history of pains culminating in free perforation (*Cases* 2, 4, 5, 6, 8); and one in which there are many inflamed diverticula and peridiverticulitis, characterized by a long history of bowel irregularity and pain culminating in perforation of a mesocolic or pericolic abscess (*Cases* 1, 3, 7, 9). The former syndrome is to be diagnosed from perforated appendicitis and peptic ulcer, the latter from perforated carcinoma.

Criteria for Resection in this Series.—In general, resection was performed upon those patients who seemed fit for it and in whom it was technically feasible. 'Fitness' meant the absence of any other serious disability and a history of robust health before onset. 'Technically feasible' meant an apparently localized area of diverticulitis in a mobile sigmoid colon. Obviously coincident intestinal obstruction contra-indicated resection in *Case* 1, and resection was not attempted in the 2 cases of advanced general peritonitis (*Cases* 4 and 6) with gross distension of the small bowel. Distension, in fact, was the chief contra-indication to resection and anastomosis. The patients with diabetic coma (*Case* 4) and long-standing disabling ankylosing spondylitis (*Case* 8) were 'unfit'. *Case* 2 fulfilled these criteria, but resection had not been considered at that time.

Results of Resection in these Cases.—One patient has no symptoms thirteen months after operation. Two have very minor symptoms fifteen and twelve months after operation. One has had two severe attacks of colic in the past six months, attributed by her to certain foods. However, eight months after resection, she says she feels well and is much less constipated than before operation. On the other hand some diverticulitic bowel was apparently left in situ in her case. It seems likely that the minor symptoms complained of in these 3 cases can be controlled by dietary measures (Welch and others, 1953).

All patients, incidentally, recovered quickly after their operations though 3 had wound infections and one of these has an incisional hernia.

DISCUSSION

1. **Resection.**—Though all agree that resection alone cures diverticulitis, opinions vary about the amount of bowel to be removed. Telling and Gruner (1916) and now Colcock (1956) have advocated removal of all diverticulum-bearing gut. However, Telling and Gruner point out that the site of election for diverticula is the sigmoid flexure and Guy and Werelius (1952) state the well-known truth that 85 per cent of those diverticula which become complicated are confined to the sigmoid.

Other surgeons (Lockhart-Mummery, 1938; Smithwick, 1942; Welch and Rodkey, 1956) find that excision of the sigmoid loop cures the disease although some diverticula are left behind. At any rate it seems essential to remove all the diverticulitis, generally 10–20 cm. of bowel. Results are poor if 'diverticulitic', as opposed to 'diverticulotic', gut remains in situ. Apart from further complications of the infected diverticula, stricture may follow the use of inflamed gut in the anastomosis (Welch and Rodkey). In Smithwick's series the average amount removed was 13·6 cm.; in Boyden's (1950) 14·5 cm.; in the present series 16·0 cm.; and in Welch and Rodkey's 20 cm. Long-term follow-up of cases of sigmoidectomy shows that there is no late mortality (Lockhart-Mummery; Smithwick; Boyden; Lloyd-Davies; Welch and others, 1953). It seems probable, therefore, that sigmoidectomy will cure most cases, minor symptoms afterwards being of little account and no danger to life.

2. **Emergency Resection.**—The best reason for emergency resection is the unsound and uncertain basis of other local measures (*see above*). However, except in cases of 'gangrenous sigmoiditis', colostomy with 'closure' and/or drainage should save life. There are no statistics to prove one method better than the other, though in the absence of an anastomosis, both should be equally safe. If, after resection, the lower end cannot be brought out, there is no reason why it should not be temporarily closed, as in Hartmann's operation.

3. **Emergency Resection and Anastomosis.**—The simple objection to emergency resection and anastomosis is that it ought to be unsafe. One is anastomosing unprepared large bowel in ill patients suffering from general peritonitis. On the other hand this bald statement turns out to be less formidable than it sounds. Resection and anastomosis of bowel for perforated diverticulitis is no innovation. The first successful case was described by Souttar in 1914, though this was a cæcal diverticulum and the operation a right hemicolectomy.

Again, as Lazarus (1933) points out, some patients with peritonitis and free perforation are not so ill as others. Perhaps this has to do with the amount of fæcal contamination.

In any case there seems no reason why peritonitis should interfere with anastomotic sutures any more than it does with the purse-string suture burying an appendix stump, or with the sutures closing a perforated ulcer. Of course if the peritonitis seems likely to be lethal of itself (gross distension and ileus, pockets of pus, peripheral circulatory failure) resection and anastomosis are pointless. But an early peritonitis ought to get better once its source is completely removed.

The safety of large bowel anastomosis depends particularly on intrinsic factors—absence of tension, proper blood-supply, non-strangulating sutures, a complete peritoneal coat. There is no reason why all these conditions should not be fulfilled at an emergency operation. Blood-supply in diverticulitis resection is in particular less endangered than in cancer surgery, where vessels must be tied more proximally.

Most surgeons would regard the absence of bowel preparation as the chief objection to this treatment. Yet if the anastomosis is made in such a way that the mucosa is properly turned in, the important healing layer (the serosa) should not be affected by bowel content at all, having no contact with it. As to mucosal healing, the contents of diverticulitic gut are not abnormal unless there has been obstruction with excessive multiplication of organisms. (In carcinoma there is both ulceration and obstruction and quite likely an excessive abnormal bacterial population; bowel preparation is theoretically desirable in cancer surgery and in practice has improved results.) Indeed, the chief place of

anti-bacterial drugs is in control of the peritonitis. There is no need to ' sterilize ' the bowel—its contents are those to which the mucosa, itself normal, is accustomed. Safe anastomosis ought to be possible in perforated diverticulitis in the absence of obstruction or otherwise lethal circumstances, e.g., an already far-advanced peritonitis.

CONCLUSIONS

1. Perforated diverticulitis is common and may be expected to become commoner in an ageing population.

2. Observation in this small series suggests that there are two fairly distinct syndromes of perforating diverticulitis, one characterized by a short history without bowel symptoms, and one or several diverticula, the other by a long history of bowel symptoms with peridiverticulitis.

3. Sigmoidectomy cures diverticulitis if care is taken to remove all ' diverticulitic ', as opposed to ' diverticulotic ', bowel.

4. Certain cases of perforated diverticulitis (' gangrenous sigmoiditis ') demand resection, and in most ' closure ', ' excision ', or drainage are theoretically unsound.

5. Emergency sigmoidectomy and anastomosis for perforating diverticulitis is safe if the patient has been well, the sigmoid colon is mobile, and there is no intestinal obstruction. It is not worth while in advanced general peritonitis.

6. Thus some cases of perforated diverticulitis can be cured by an operation which saves life.

SUMMARY

The history of the management of perforated sigmoid diverticulitis has been described, its pathological basis re-examined, and the place of radical surgery in it discussed with reference to 9 cases operated upon by the author from 1954 to 1956.

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PATENT VITELLO-INTESTINAL DUCT

THE RISK OF OBSTRUCTION DUE TO PROLAPSE

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COMPLICATIONS associated with vitello-intestinal remnants are many and varied, but the patent vitello-intestinal fistula is one of the rarest. This paper is based on experience with 4 babies with vitello-intestinal fistula, in whom prolapse through the fistula led to intestinal obstruction. We recommend early operation on such fistulæ to prevent this serious complication.

EMBRYOLOGY

The yolk-sac of the human embryo makes its appearance about eleven days after fertilization; three weeks later, the vitello-intestinal duct which joins it to the primitive gut has largely shrivelled and gone, though the sac may persist for some time longer. Its function in relation to the developing embryo is as mysterious as the brevity of its existence. The presence of primitive blood-cells in its walls has suggested that it may play some part in early hæmopoiesis, but it is more likely that the sac is simply a vestigial remnant of a primitive ancestry common to birds and mammals.

FIG. 724.—*Case* 1. Retrograde intussusception of vitello-intestinal duct.

The commonest form of persistence of the duct is the Meckel's diverticulum whose apex lies free in the peritoneal cavity. It may be quite innocuous or it may give rise to trouble because of peptic ulceration in heterotopic mucosa within its walls, leading to bleeding or perforation.

In a second and much less common form of persistence of the duct, its apex opens at the umbilicus. As a rule the only disability is a slight mucous discharge, though occasional torsion of the duct occurs (Gross, 1953). Occasionally a bright red protruberance appears at the umbilicus after separation of the cord, the so-called entero-teratoma.

In the 4 cases here recorded the duct underwent prolapse and by dragging loops of small bowel into it, caused intestinal obstruction and in one case necrosis of the gut.

CASE REPORTS

Case 1.—A male, full-time, child was born in the Maternity Department, Dundee Royal Infirmary, on Nov. 16, 1950. After a first-stage labour lasting 43 hours, signs of fœtal distress with passage of meconium and cardiac irregularity had appeared. Haig Ferguson forceps were applied and a mid-cavity extraction performed without difficulty. The child cried at birth and his general condition appeared satisfactory. A small cystic swelling was noted at the base of the umbilical cord and from this a red bifid structure protruded, and lay on the anterior abdominal wall (*Fig.* 724). The appearance was likened by a member of the nursing staff to the uterus and Fallopian tubes. Closer inspection showed a T-shaped structure some 5 in. in length with a red congested mucosal covering. The fœtal end of this was enveloped in a thin opaque membrane, to the caudal surface of which was attached the umbilical cord. At the ends of the horizontal limbs of the T there was a dimple-like depression. A probe introduced into each revealed a lumen passing along both limbs and through the umbilicus. The exact nature of the lesion was not immediately apparent, but it was clear that there was prolapse of bowel through some congenital defect of the umbilicus, and after pre-operative preparation, operation was undertaken approximately 6 hours after birth. An elliptical incision was carried round the base of the extrusion close to the junction of skin and membrane and deepened to the peritoneum. A small vertical incision was then made in the membrane, which was reflected laterally, disclosing the presence of two parallel loops of bowel emerging from the peritoneal cavity and passing into the mucosal covered segment of the protrusion (*Fig.* 725). On opening the peritoneal cavity, proximal and distal coils of terminal ileum were seen

FIG. 725.—*Case* 1. Detail of the anatomical disposition of the intussusception as exposed at operation. The lower part of the everted vitello-intestinal duct and the two coils of intestine entering into it can be seen. The ligated cord lies on the right.

drawn up to the under aspect of the umbilicus and intussuscepted through it. The junction of the membrane and mucosa formed a tight ring which prevented reduction of the prolapsed bowel, but after the vertical incision had been carried distally through this ring and half an inch into the gut distal to it, it was possible to draw the whole protrusion into the abdominal cavity. It then became obvious that two intussusceptions had occurred through a patent vitello-intestinal duct. These were now reduced without difficulty and both proved viable.

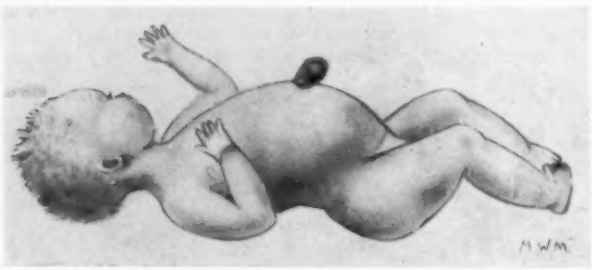

FIG. 726.—*Case* 2. The so-called entero-teratoma, a protrusion of intestinal mucosa at the umbilicus seen 1 week after birth.

FIG. 728.—*Case* 2. Detail of the degree of retrograde intussusception. A window has been taken out of the prolapsed vitello-intestinal duct and the two coils of bowel entering into it and being compressed can be seen.

The duct was excised and the defect in the antemesenteric border of the bowel-wall closed with a double layer of catgut. A large defect was present in the mesentery of the small bowel. Post-operatively the child made good progress, and the bowel moved normally on the third day. He left hospital on Dec. 5, 1950, having regained his birth-weight.

Ten days later he was re-admitted from the postnatal clinic with a history of increasing abdominal distension and vomiting of some twenty-four hours' duration. Radiographic examination showed distension of the small gut with scattered fluid levels. The abdomen was re-opened on Dec. 16. The terminal ileum and cæcum were found to be involved in a diffuse plastic peritonitis with multiple adhesions and pockets of pus lying between the involved coils of gut. The ileum above the site of obstruction was grossly distended. An ileo-transverse colostomy was performed to by-pass the site of obstruction and the free pus swabbed out. Despite vigorous measures to maintain fluid and electrolyte balance the child died the following day. Permission for a post-mortem examination was not obtained.

Case 2.—A male child, after a full-time normal delivery, was noticed to have a bright red protuberance at the umbilicus after the cord remnant had separated. He was seen at the surgical out-patient department at

FIG. 727.—*Case* 2. Prolapse of the whole of a patent vitello-intestinal duct five weeks later with symptoms of intestinal obstruction.

Forfar Infirmary when 1 week old. It was then noted that the protuberance was 1 in. in length, bright red in colour, and resembling intestinal mucosa. At its tip there was a small dimple from which a small amount of mucus was exuding (*Fig.* 726). A diagnosis of patent vitello-intestinal duct was made, but since the bowel motions were normal and the child was reported to be thriving, it was decided to defer operation for 2–3 months. This proved to be the wrong decision because the child was admitted to hospital 5 weeks later with intestinal obstruction. Examination of the abdomen revealed an extraordinary state of affairs. A single barrelled loop of everted intestine with an aperture at its tip was protruding from the umbilicus (*Fig.* 727). The loop was dark purple in colour and measured 5 in. in length. The child's abdomen was distended; all feeds had been vomited, and no stool had been passed for 48 hours. The eyeballs and fontanelles were retracted from dehydration.

A pre-operative diagnosis of intestinal obstruction associated with prolapse of a patent vitello-intestinal duct was made and preparations for operation were proceeded with. The stomach was emptied by catheter, and a fine polythene tube was inserted into the saphenous vein through which 100 c.c. of blood, followed by a slow infusion of 5 per cent dextrose in water was given.

Operation was begun by incising the single barrelled loop and demonstrating that the condition was a retrograde intussusception (*Fig.* 728). Two loops of collapsed ileum, afferent and efferent, were contained in the duct. The umbilicus was encircled with an incision through all layers, and this was extended into a right paramedian incision. The patent duct, together with its contained small bowel, was then delivered to the exterior via the paramedian incision. When the parts were inspected it was apparent that the duct and the bowel immediately adjacent to it were so devitalized by strangulation that resection was necessary. After this was done, continuity was re-established by end-to-end anastomosis. To be quite certain about the patency of the stoma in this young baby, an entero-anastomosis was carried out between efferent and afferent loops.

The child made a satisfactory convalescence and was discharged 8 weeks after operation.

Case 3.—A male child, after a full-time, normal delivery, was referred to the Royal Hospital for Sick Children, Glasgow, at 6 days because of a heavy odour

from the sloughing umbilical cord and the appearance of herniation of the bowel.

On examintion, the umbilical cord was thick and infected and protruding from its end there was a red mass resembling the prolapse of mucosa through a colostomy. There was a small central opening discharging greenish material and a few bubbles of gas. Since the child was in good condition he was given a course of chlortetracycline and kept under observation.

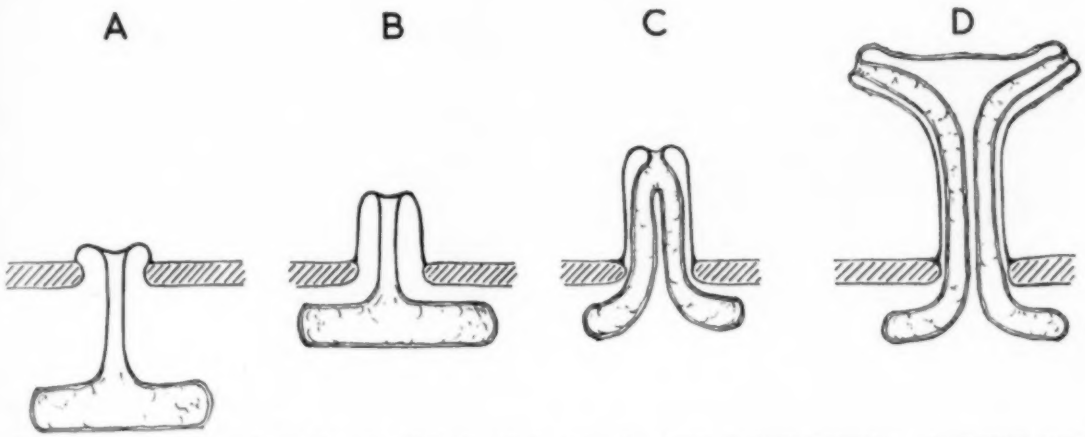

FIG. 729.—Degrees of prolapse of patent vitello-intestinal duct. A, Simple mucosal protrusion. B, Further stage of prolapse. C, Complete prolapse of the duct with small bowel dragged into it. D, Prolapse of duct and coils of intestine adjacent to it.

Ten days later he began to vomit and the prolapse of bowel at the umbilicus became worse until 4 cm. of bowel were exteriorized. A decision to operate was made and under general anæsthesia the abdomen was opened through a right rectus incision. A long patent vitello-intestinal duct was found prolapsing through the umbilicus and presumably kinking the small intestine. The base of the diverticulum was clamped and the ileum closed transversely. The remains of the diverticulum were everted through the umbilicus and excised. The child made an uneventful recovery.

Case 4.—A male child, aged 7 days, was admitted to the Royal Hospital for Sick Children, Glasgow, with vomiting and prolapse of a reddish mass at the umbilicus, with some bubbles exuding when the baby cried.

On examination at the hospital, the cord was separating and 1 in. of red granulating bowel with a small central opening was protruding from the umbilicus. The diagnosis of a patent vitello-intestinal duct was made, but since the child was well, it was decided to treat it expectantly. However, 8 days later the baby began to vomit and the bowels stopped moving. A further protrusion of the bowel occurred at the umbilicus and the decision to operate was made.

Under general anæsthesia, the abdomen was opened through a right paramedian incision. A long patent vitello-intestinal duct was discovered passing from the ileum into the umbilicus. Its base was clamped opposite the ileum and the latter closed transversely in two layers. The severed fistula was everted through the umbilicus and removed. The child made an uneventful recovery.

DISCUSSION

The principal point of interest in these 4 cases is the progressive eversion of the duct with the development of intestinal obstruction. Three processes may be involved in its production.

First, raised intra-abdominal pressure during coughing, crying, straining, or during periods of fœtal distress, tend to propel the intra-abdominal part of the duct through the umbilicus. The mechanism is exactly similar to a prolapse of a colostomy stoma.

The second factor is probably the muscular power of the gut wall itself once eversion has begun; peristalsis and antiperistalsis continue the process of eversion until the duct is turned completely inside out (*Fig.* 729 C). Once this has happened the two loops of ileum are dragged into the sac formed by the everted duct. If the process continues, a bicornate projection appears at the umbilicus, as in *Case* 1 (*Fig.* 729 D). Alternatively, the condition may be seen before this stage is reached, when a single loop is present. (*Fig.* 729 C.)

A third factor may be the presence of the obliterated vitelline vessels which pass from the duct across the ileum to gain attachment to the mesentery. As eversion and prolapse of the duct proceed, this fibrous cord may help to pull the ileum into the duct and so form the apex of an intussusception.

When retrograde intussusception has taken place, resection of the duct, and if necessary the affected segment of bowel, is the treatment of choice.

When a child is seen with a symptomless protruberance of intestinal mucosa at the umbilicus, what procedure should be adopted? If the intra-abdominal part of the duct is contracted and fibrosed, and only the terminal part is patent, little danger is to be anticipated by waiting until the child is older (Howard, Moss, and O'Domhnäill, 1953). If, however, the duct remains patent throughout its whole extent there is a risk of simple prolapse, (*Cases* 3 and 4), leading, if the process continues, to intussusception and possible necrosis of the bowel. Because of these risks it would appear wiser to explore the abdomen early in all babies who show intestinal mucosa at the umbilicus. To resect a persistent vitello-intestinal duct in a healthy child is one thing; it is a very different matter to resect a gangrenous segment of gut in an ill and dehydrated child who has suffered from intestinal obstruction for some days.

From a study of the literature it is clear that the condition is rare. Moore and Shumaker (1952) note

that until 1952, 35 cases were recorded with 4 recoveries. Aitken (1953), reporting the experience only of vitello-intestinal duct remnants at the Royal Hospital for Sick Children, Glasgow, between 1932 and 1952, records no case of retrograde intussusception through the umbilicus. No examples of this condition are recorded in the series of one hundred and forty nine cases of anomalies of the vitello-intestinal duct described by Gross (1953).

SUMMARY

1. Four cases of intestinal obstruction in newborn children due to the prolapse of a patent vitello-intestinal duct are recorded.

2. Three stages of the prolapse may be recognized. In the first, the duct only is everted. In the second, ileum is dragged into the duct, and in the third, duct and ileum are everted. Examples of each of the 3 stages are described.

3. All cases were treated by resection of the duct. Three of the babies survived.

4. The authors advocate early exploration of the abdomen of children with prolapse of intestinal mucosa at the umbilicus to prevent this serious complication.

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CARCINOMA OF BREAST ARISING IN A FIBRO-ADENOMA

BY H. S. KELLETT AND F. R. R. MARTIN, BRADFORD

MALIGNANT change arising in a fibro-adenoma of the breast is a rare event: when it occurs, it almost always takes the form of a sarcoma (Willis, 1953). A carcinoma developing in a fibro-adenoma is a histological curiosity. Very few undisputed cases are reported in the literature, and probably none prior to 1949. The following case is therefore recorded in view of its rarity.

CASE REPORT

Mrs. A. T., aged 55, was referred to the Surgical Out-patient Department of St. Luke's Hospital in May, 1954, complaining of a swelling in her left breast. She said the tumour had only been present for the last two months and she thought it had increased in size in the last two weeks. Her previous medical history contained nothing of note except that in 1952 she was treated in the Gynæcological Department of St. Luke's Hospital on account of post-menopausal hæmorrhage: dilatation and curettage was performed, but failed to show any cause for this complaint. No tumour in the breast was noted at this time in her case history.

On examination, a curiously shaped, lobulated swelling, some 4 cm. in length, was detected in the upper and outer quadrant of the left breast. Though the swelling appeared to be well defined and mobile, the overlying skin was adherent to it. The tumour was not attached to the underlying fascia or muscle and was easily palpable with the flat of the hand. There were no palpably enlarged glands in the axilla.

A diagnosis of probable carcinoma of the breast was made and on June 5, a simple mastectomy was performed.

The pathological report (*see below*) showed the presence of a carcinoma arising in a fibro-adenoma of the breast, with no evidence of infiltration of the surrounding breast tissue.

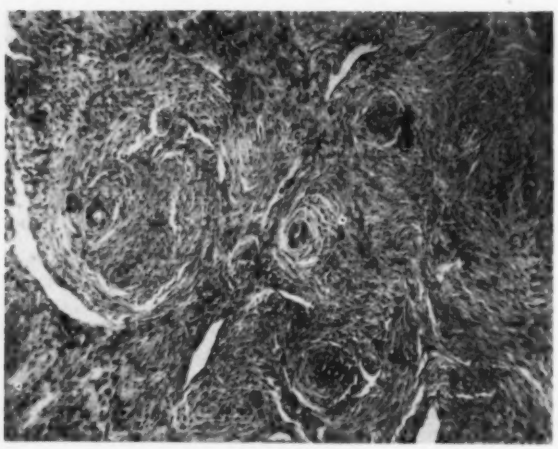

FIG. 730.—Area of tumour showing pericanalicular fibro-adenoma. (× 25.)

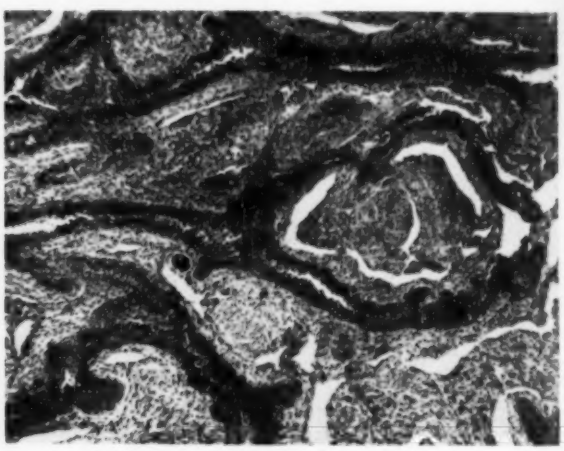

FIG. 731.—Area with intracanalicular structure. (× 52·5.)

Following the receipt of this report the question of radiotherapy was discussed. It was decided, however, that none should be given unless signs of local metastases arose.

FOLLOW-UP.—The patient was seen at three-monthly intervals. In January, 1955, a radiograph of chest showed no abnormality, but in April, 1956, she had a hæmoptysis. Her general condition had deteriorated though there were no signs of local recurrence of the tumour.

A further radiograph of the chest showed evidence of numerous metastases, and two swellings (presumably metastases) were detected on the left iliac crest. Death occurred in May, 1956, almost two years after operation.

Pathological Report.—"In the upper and outer quadrant of the breast there is a discrete lobulated firm tumour measuring 4 × 3 × 3 cm. It is bounded medially by breast tissue, and laterally by adipose tissue. The remaining breast is of fibrous consistency and contains a few small cysts. In another quadrant there is a second discrete fibrous nodule of 1 cm. diameter.

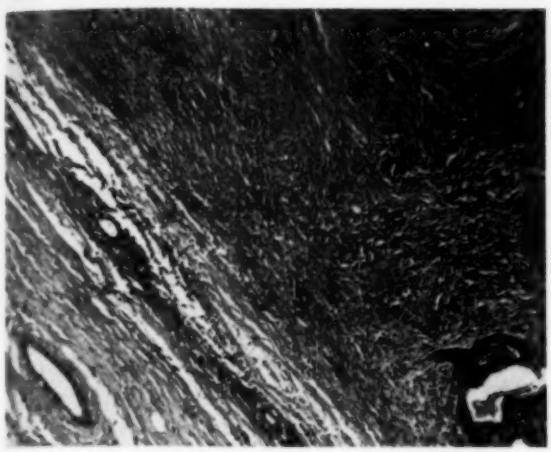

FIG. 732.—Cellular margin of tumour bounded (bottom left) by fibrous breast. (× 52·5.)

FIG. 733.—Area of hyperplastic glandular clefts. (× 52·5.)

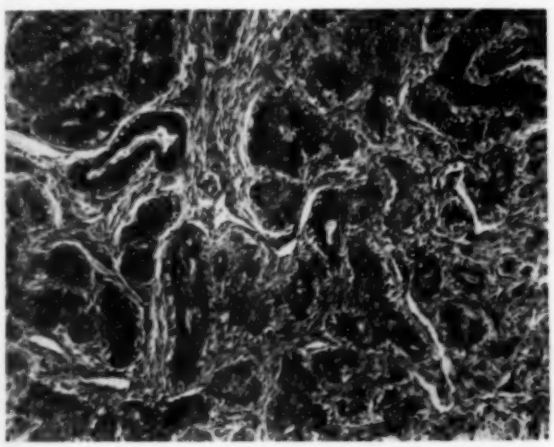

FIG. 734.—Adenocarcinomatous field in breast tumour. (× 52·5.)

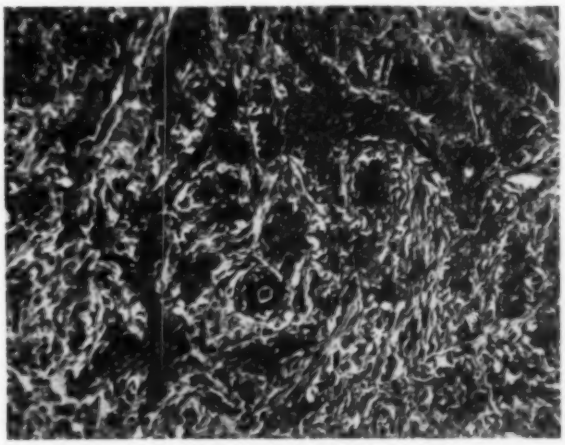

FIG. 735.—Anaplastic carcinoma infiltrating the stroma. (× 52·5.)

"The main tumour has the general structure of a cellular fibro-adenoma, with clefts lined by hypertrophic epithelium. Although in some places the pattern is peri-canalicular, in the main the tumour presents an intra-canalicular appearance (*Figs.* 730, 731). One section shows an almost purely fibroplastic picture, with only scanty glands, but is sharply delineated from the tissue surrounding the tumour by its greater cellularity (*Fig.* 732). Nine other sections all exhibit various stages in transition from a benign tumour to a carcinoma. Many areas present an intermediate picture, in which the glandular clefts are lined by cuboidal epithelium of several layers in thickness, and their lumina contain desquamated round cells with small pyknotic nuclei and foamy cytoplasm (*Fig.* 733). In these areas the hypertrophic epithelium also forms buds invading the fibrous tissue, but mitotic figures are scanty. In other fields there are both frank adeno-carcinoma and solid columns and trabeculæ composed of anaplastic spheroidal cells (*Figs.* 734, 735). The connective-tissue component of the tumour is very cellular and stains weakly for collagen fibres compared with the remaining breast tissue. There is a varying admixture of lymphocytes, and in places a few carcinoma cells infiltrate between the fibroblasts. In one area it is impossible to distinguish with certainty between individual elongated epithelial and stromal cells, but there is insufficient evidence to suggest a concomitant sarcomatous change. The tumour is surrounded by a false capsule of condensed fibrous breast tissue except on the lateral side, where it abuts on adipose tissue. In the latter region there are a few fat-laden cells and giant-cells of foreign-body type.

"Section of the other breast nodule shows a simple fibro-adenoma of usual structure. Several sections taken from other parts of the breast all show simple fibrocystic hyperplasia and reveal no evidence of malignancy elsewhere."

DISCUSSION

Four out of 302 cases of carcinoma reviewed by Rose (1942) were thought to have originated in fibro-adenomas, but are insufficiently documented. Smith (1949) illustrates an intraduct carcinoma arising within an intracanalicular fibro-adenoma. Photo-micrographs of a comedo-carcinoma developing in a hyalinized intracanalicular fibro-adenoma are recorded

by Stewart (1950), who comments on its extreme rarity. A third case, described by Austin and Fidler (1953), is a focus of poorly-differentiated carcinoma in a hyalinized intracanalicular fibro-adenoma and confined within its false capsule. Hill and Miller (1954) report a series of 'pure' adenoma of breast, in one of which arose a carcinoma. Stout (1954) stated that he had not seen an example of carcinomatous change in a fibro-adenoma of the breast until that year.

There are a few records of composite tumours which have probably developed in fibro-adenomas. Tudhope (1939) reported a very unusual complex tumour with fibro-adenomatous, carcinomatous, sarcomatous, osteoid, and cartilaginous areas, in which the sarcoma and carcinoma were probably formed by malignant changes in both elements of the fibro-adenoma. Another composite tumour consisting of carcinoma and osteogenic sarcoma recorded by Budd and Breslin (1937) may have arisen in an old fibro-adenoma as there was a history of breast tumour for twenty-eight years. A 'carcinosarcoma' described by Cronkite, Dockerty, and Waugh, (1949) was thought to have originated in a fibro-adenoma.

Cheatle and Cutler (1931) and Willis (1953) draw attention to examples of invasion of a fibro-adenoma by malignant cells from a neighbouring carcinoma, an event which could lead to a false opinion on the histogenesis. We have ourselves recently encountered such a case. This possibility of carcinomatous invasion of a fibro-adenoma was excluded by serial sections of the intervening breast tissue in the case described by Smith (1949), but may account for spurious reports in breasts which have been less carefully examined.

Experimentally Selbie (1942) obtained a carcinomatous change in a transplantable fibro-adenoma in rats. A carcinoma appeared in one transplantation line from a spontaneous tumour found in 1934. Previous lines had given rise frequently to sarcomas '*en passage*'. The carcinomatous change appeared first in a male animal and was irreversible, further examples of carcinoma and carcinoma in fibro-adenoma being obtained on transplantation in both male and female rats after long intervals. The author suggested that the powers of proliferation of the epithelium overcame the restraint of the fibrous component of the tumour.

It is noteworthy that the three previously recorded clinical cases (Smith, 1949; Stewart, 1950; Austin and Fidler, 1953) all arose in old hyalinized intracanalicular fibro-adenomas. In the present case the connective-tissue component of the tumour was much more cellular, resembling that of a 'giant fibro-adenoma' or 'cystosarcoma phyllodes'. The parent tumour appeared to be of recent origin, the nodule being first noticed by the patient two months previously and not recorded in an examination two years earlier. Thus the tumour probably developed three years after menopause, an occurrence, however, which is not uncommon in the cellular type of intracanalicular fibro-adenoma. If any comment is to be made on the clinical features of this case, it is that when the patient was first seen, considerable doubt was experienced as to the exact diagnosis as indicated by the physical signs. This doubt would appear to find substantiation in the pathological report on the tumour.

SUMMARY

A case of carcinoma of the breast arising in a fibro-adenoma is described. The rarity of this condition is stressed.

We wish to thank Professor R. A. Willis and Dr. F. E. Chester Williams for their kind interest and advice in this case, and Mr. P. Harrison for the photomicrographs.

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REVIEWS OF BOOKS

Additional Reviewers are required for French and German books. Such books would become the property of the Reviewer. Surgeons interested should communicate with the Editorial Secretary.

TRAUMATIC ŒSOPHAGEAL PERFORATION

REPORT OF THREE CASES

By RONALD R. GILFILLAN

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EACH of the following cases presents important aspects of a rather uncommon condition. The first may well be unique, but the other two should be considered in conjunction with approximately 100 œsophagoscopies performed without mishap.

CASE REPORTS

Case 1.—A 2-year-old female child was brought to the Surgical Out-patient Department of Dundee Royal Infirmary at 5 p.m. on July 11, 1956, with a somewhat sketchy history of having fallen from a first-floor window onto a flower bed. She was picked up by a neighbour who noticed a length of garden cane protruding from the collar of her dress. This was withdrawn. No resistance was appreciated and the few specks of blood on the cane were attributed to a tiny wound in the right side of the neck about 2 cm. above the mid-point of the clavicle.

When seen at this time in the casualty department the child was fretful and crying, but did not seem to have been severely injured. The wound was quite clean and healthy, and undamaged muscle tissue was seen beneath. A.T.S. and penicillin were given, the wound was sutured, and the child was sent home.

Two hours later the child was brought back to the hospital, having in the interim complained of pain in her back. On examination there was moderate surgical emphysema of the face, neck, and chest. Respiration was rapid but not laboured, and there was slight peripheral cyanosis. Breath-sounds were audible in the right chest but were appreciably fainter than those on the left. P.A. chest film showed a partial right pneumothorax and a small left pleural effusion. The overlying subcutaneous emphysema obscured any emphysema that might have been visible along the mediastinal tissue planes.

The child was then screened, and lipiodol given by mouth demonstrated leakage low in the posterior mediastinum. Despite the high level of the skin wound, no evidence of injury to the cervical œsophagus was found.

At 11 p.m. a right thoracotomy was made through the bed of the 4th rib. The right pleural cavity was heavily contaminated with frothy, brown, watery fluid and the entire pleural surface showed marked inflammatory reaction. The wound was seen to pass through the apex of the right pleural cavity just anterior to the right subclavian vein, without having damaged the right upper lobe, down posterolateral to the superior vena cava and medial to the vena azygos, excoriating but not rupturing the pericardium overlying the right atrium, anterior to the lower pulmonary vein, and apparently ending in a spiral tear 4–5 cm. long through all coats of the œsophagus. Forward displacement of the œsophagus at this level revealed that the left pleura was likewise contaminated. An attempt was made to palpate the dome of the left diaphragm, but no lesion could be felt here. The œsophagus was sutured with a double layer of interrupted linen sutures and the mediastinal pleura repaired over it. An intercostal drain with underwater seal was inserted in the 8th interspace laterally and the wound was closed in layers. The left pleural cavity was similarly drained at the conclusion of the operation.

Antibiotic therapy was continued during the postoperative period with penicillin and streptomycin. Complete pulmonary re-expansion was confirmed radiologically. General condition appeared to improve slightly thereafter until some 4 hours later (approximately 4.30 a.m.) when the blood-pressure, which had been well maintained, began to fall and despite resuscitative measures the child died at 8 a.m.

At necropsy on the following day there was no evidence of leakage around the œsophageal repair, but the cupola of the left diaphragm presented an area of bruising immediately subjacent to which lay a perinephric hæmatoma attributable to a 1-cm. laceration on the upper pole of the left kidney.

Comment.—Several aspects of this case seem to merit further comment:—

1. The misleading appearance of normal muscle beneath the skin wound due to these tissues resuming their normal relationship to one another on withdrawal of the cane.

2. During the actual screening it was noted that the escape of lipiodol into the peri-œsophageal areolar tissue was difficult to observe—indeed, had it not been that films were taken in addition, it is conceivable that the leak might have passed unnoticed altogether, since the site of the external wound directed attention largely to the cervical œsophagus. These films formed the crux of the diagnosis as the surgical emphysema could have resulted from perforation of, say, lung or trachea.

3. The wide infected area for 6–7 hours prior to operation rendered the fatal outcome a foregone conclusion, but it is difficult to see how this delay could have been obviated in view of the paucity of clinical signs when the child was first seen.

Case 2.—A 69-year-old male was first seen at 2 a.m. on Feb. 25, 1956, having undergone œsophagoscopy at 3 p.m. on the previous day. The examination was negative but moderate bleeding at the lower thoracic œsophagus was seen on withdrawal of the instrument. He complained of pain in the neck and lower left chest since 7 p.m. when slight emphysema of the face and neck was observed. He was transferred from the peripheral hospital (25 miles distant) to Dundee Royal Infirmary.

By this time there was gross surgical emphysema of the face, neck, and trunk—also extending down the arms as far as the wrists, and down the thighs to the knees. Dyspnœa was marked (respiratory rate about 36 per minute) with peripheral cyanosis; the pulse was rapid and feeble. The systolic blood-pressure was 80 mm. Hg. The diastolic level was unobtainable. A straight P.A. film of the chest showed partial atelectasis of the left lower lobe, but further detail was obscured by the gross surgical emphysema. Despite the assumption that perforation had probably occurred low in the thorax a few cc. of lipiodol was given by mouth. This leaked out into the cervical tissues at the lever of C.6, and some tracked down to the base of the left pleural cavity.

Operation was performed at 4 a.m. A transverse cervical incision was made, the infrahyoid muscles on the left divided, and the left sternomastoid displaced posteriorly. The areolar tissue around the left lobe of the thyroid and lateral aspect of the œsophagus showed a dirty dark-brown discoloration. The tear was located in the posterior wall of the cervical œsophagus at the level of the intervertebral disk between C.6 and C.7, where osteophytic lipping of marked degree was observed.

Repair was effected with a single layer of interrupted silk (in view of the patient's poor general condition, the second layer was omitted,) and the wound was closed in layers with a tube drain to the site of repair.

Post-operative film on the following day showed persistence of the left lower lobar collapse. This responded to bronchoscopic aspiration under local anæsthesia. The patient's general condition improved until the seventh day when a profuse purulent discharge issued from the site of the drainage tube (removed fourth day). This was replaced and the patient was fed by a Ryle tube for the ensuing 7 days.

A chest film on the fourteenth day revealed a fluid level at the left 6th rib posteriorly. Aspiration produced foul-smelling thick yellow pus. This posterior empyema was finally drained by rib resection on the twenty-first day following inadequate control by intercostal catheter drainage. A fortnight later the sinus had healed and chest films were satisfactory.

On May 4 a further lipiodol swallow showed a para-œsophageal cavity at the level of the previous repair. As it seemed that spontaneous closure of the fistula was unlikely or, at best, would take some considerable time, operative closure was undertaken on May 29.

The previous scar was re-opened and the dissection carried down around the sinus track, which was excised together with the para-œsophageal pouch. This consisted of fibrous tissue only. The edges of the opening into the œsophagus were freshened and a double layer interrupted silk repair was performed. The wound was then closed in layers and drainage was effected through a separate stab wound in Burns' space.

Thereafter convalescence was uneventful. When seen as an out-patient on Aug. 5 the chest film showed only residual basal pleural thickening: he felt well, had regained his normal weight, and deglutition was normal.

Comment.—The following points of interest emerge:—

1. Perforation of the thoracic œsophagus seemed certain, and the general condition of the patient so poor that a lipiodol swallow was given with some diffidence. The value of this examination cannot be emphasized too strongly as thoracotomy would not have given access to the lesion.

2. In view of the spread of lipiodol to the left pleura, subsequent rib resection could have been avoided had intercostal drainage been established at least on the first post-operative day.

3. The initial repair was probably inadequate and this was the precipitating factor in the breakdown of the wound on the seventh day, although subsequent investigation revealed that solid food had been administered inadvertently.

4. The ease with which the cervical œsophagus had been perforated by pressure between the instrument and the osteophyte at C.6, 7 is a further example of this real hazard during the examination in elderly persons (Goligher, 1948; Franklin, 1948; and Kettel, 1948).

5. The second operation was much easier than had been anticipated, and seems amply justified in respect of a reduced convalescence.

Case 3.—An 86-year-old female was seen on Jan. 18, 1957, with a history of increasing dysphagia for some two months. Only small quantities of fluid could be taken very slowly. Barium meal, performed one week previously, had demonstrated an œsophageal diverticulum of 'traction' type just below the aortic arch, without hold-up at this point, and an ulcer on the lesser curvature of the stomach close to the cardia. In view of her distress, œsophagoscopy was performed with the object of inserting a Souttar tube.

The œsophagus was full of inspissated barium and fermented food. When this had been partly cleared the anterior wall about the middle third was œdematous and congested. No pouch could be seen. Two biopsies were taken at this site. The lower half of the viscus showed marked œsophagitis, and at 38 cm. a tight stricture was appreciated. As adequate bouginage was not feasible, a further blind biopsy was taken and the instrument withdrawn. The presumptive diagnosis was carcinoma of the gastric fundus.

The patient began to complain of pain in her back some 3 hr. later. P.A. chest film revealed a 50 per cent left pneumothorax with effusion. It was considered likely that perforation had occurred at the site of bouginage.

Lipiodol swallow demonstrated leakage just below the aortic arch. As her general condition seemed good it was decided to repair the defect.

A standard left thoracotomy through the bed of the resected 8th rib was performed 5 hours after œsophagoscopy. The pleura was full of foul brownish fluid, and showed an intense generalized pleuritis. Immediately below the aortic arch lay a mass of caseating lymph-nodes, adherent to the œsophagus where perforation had occurred, through the left pulmonary ligament. The mucosal layer was friable and œdematous. The repair, by single layer of interrupted silk sutures, was 'bolstered' by suturing flaps of mediastinal pleura, and as much of the gland tissue as seemed viable, over the lesion. Further exploration revealed a small hard tumour just below the cardia. The diaphragm was opened between the lateral and posterior branches of the phrenic nerve, and the lateral aspect of the gastric fundus drawn through. The œsophagus was transected about 3 cm. above the hiatus œsophageus, the distal stump invaginated with silk sutures, and a gastro-œsophagostomy in two layers of interrupted silk was made. The wound was then closed in layers with intercostal water-seal drain.

On the following day the patient had greatly improved. The chest film showed complete re-expansion of the lung. Antibiotic therapy was continued with penicillin and streptomycin.

On the fourth post-operative day the patient suddenly collapsed, blood-pressure could not be recorded and, despite resuscitative measures, she died at 9.30 a.m.—presumably of massive pulmonary embolus. Permission for necropsy was not granted.

Comment.—The salient factors in this instance appear to be:—

1. Barium-meal examination in the presence of obvious obstruction was unwise—lipiodol would have been far safer—for the lesion was thus obscured.

2. The pouch was known to be present but it could not be localized on endoscopy. However, in retrospect, its radiological appearance together with the closely associated calcified mediastinal lymph-nodes, and the congestion noted above, might have been a more obvious warning had it not been for—

3. —the fundal carcinoma which was, surgically at any rate, the last straw. (Parma and Riberi, 1954.)

SUMMARY

Three cases of traumatic œsophageal perforation are presented. The following suggestions are offered as to management:—

1. Operative treatment should not be delayed (Aird, 1949).

2. Lipiodol swallow should never be omitted as it is the only means of sure localization. Films should also be taken since the circumstances of screening examination may be such that little time is afforded for dark-adaptation.

3. A barium meal is contra-indicated in moderate or severe dysphagia. Œsophagoscopy should precede it, or, failing this, some such medium as lipiodol

which will not obscure the endoscopic field should be used.

4. The complication rate in œsophagoscopy is nowadays very low, but it would seem that the examination is preferably undertaken where adequate facilities are readily available.

5. Conservative management is seldom successful.

I wish to thank Mr. G. M. Sturrock for his permission to treat these cases in his units at Dundee Royal Infirmary and at Arbroath Infirmary.

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INFARCTION OF ILEUM FOLLOWING RESECTION OF COARCTATION OF THE AORTA

BY H. C. REID AND RUSSELL DALLACHY

FROM THE SURGICAL WARDS AND CLINICAL LABORATORIES OF THE VICTORIA INFIRMARY OF GLASGOW

MUCH progress has been made in the surgical treatment of coarctation of the aorta since the pioneer work of Crafoord and Nylin (1945) and Gross and Hufnagel (1945). At the present day operative correction of coarctation in patients with no associated cardiac anomalies has become a routine procedure with a mortality rate which compares favourably with that of other major surgical operations.

The earlier complications were related to the technique, but in 1953 Sealy, of North Carolina, assessing a series of 22 cases, first drew attention to an interesting abdominal complication met with in one of his cases. This patient, a few days after the operation for coarctation of the aorta, developed severe abdominal pain and died on the tenth post-operative day. Autopsy revealed diffuse arteritis affecting arteries of muscles and viscera distal to the site of coarctation. Five similar cases have since been described in the literature (Lober and Lillehei, 1954 (2 cases); Perez-Alvarez and Oudkerk, 1955; Benson and Sealy, 1956; Ring and Lewis, 1956).

The case reported here had many clinical and pathological features in common with those previously recorded and though microscopically the arterial changes were significantly different, it is believed that as a complication of resection of coarctation of the aorta the aetiology is the same.

CASE REPORT

D. P., a male child 8 years of age, was admitted to the Thoracic Unit at Mearnskirk Hospital on Sept. 24, 1956, for resection of a coarctation. His birth and infancy had been normal. At the age of 5, while convalescing from scarlet fever, he was found to have a systolic precordial bruit and absent femoral pulses. On subsequent investigation coarctation of the aorta, complicated by active rheumatic heart disease, was diagnosed.

ON EXAMINATION.—A thin pale child, fairly tall for his age. No evidence of over-development of 'upper half'. No cyanosis present. The pulse-rate was 114 per minute and regular. Apex beat was visible in the 6th left interspace 3 in. from the midline. Blood-pressure in both arms was 142/82 mm. Hg. In the lower limbs no pressure was recordable and neither abdominal nor femoral pulsation was palpable.

Radiographic examination of the heart and great vessels showed an increase in the size of the left ventricle; this was particularly evident in the left anterior oblique view during a barium swallow. The aortic arch was traceable in the postero-anterior and oblique views. Rib notching was absent.

The cardiogram showed splintering of the QRS complex in Lead 3 and slight left preponderance in the chest leads. The phonocardiogram showed the presence of an early systolic murmur at the aortic area and a short mid-diastolic bruit at the mitral area.

The sedimentation rate (Westergren's method) was 40 mm. in the first hour. C-reactive protein was not present.

FIRST OPERATION (Mr. R. S. Barclay and Mr. T. Welsh).—On Oct. 2, under hypothermia anæsthesia, left lateral thoracotomy was performed below the lower edge of the 5th rib. The aortic coarctation was demonstrated and resected. A full-width end-to-end anastomosis was made using a continuous everting suture. There was no bleeding from the anastomosis when the clamps were released. The operation lasted a little over 2 hours.

POST-OPERATIVE COURSE.—The body temperature had returned to within normal limits in 5 hours. Progress was satisfactory until the eleventh post-operative day when the patient complained of colicky abdominal pain and vomited some bile-stained fluid; these symptoms were transient, but recurred next day. On the thirteenth post-operative day the pain became constant and was located in the lower abdomen. Vomiting persisted; oral fluids were stopped and intravenous fluids begun. Temperature and pulse were normal. The tongue was clean but dry. There was some tenderness in the lower abdomen but no muscle guarding. Rectal and urine examinations revealed no abnormality. The white-cell count was 8000 per c.mm.

On the fourteenth day post-operatively the temperature was 99·8° F. (36·9° G.) and the pulse 128 per minute. The pain appeared more severe and was aggravated by movement. There was a well-defined area of extreme tenderness and muscle guarding just below and to the left of the umbilicus. Rebound tenderness was marked. The white-cell count was now 25,000 per c.mm. The child was transferred to the Victoria Infirmary, Glasgow.

SECOND OPERATION (H. C. R.).—On Oct. 16 the abdomen was explored through a right rectus-splitting incision. When the peritoneal cavity was opened copious turbid fluid escaped. The omentum was adherent to a loop of ileum about 60 cm. from the ileocæcal valve. On detaching the omentum the underlying bowel was reddened and œdematous over a linear extent of 30 cm.;

FIG. 736.—The portions of the excised segment of ileum showing irregular areas which are lustreless and which tend to be placed towards the antimesenteric border. (After fixation.) (× approx. ½.)

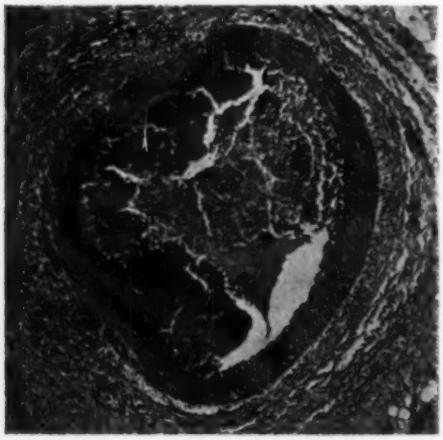

FIG. 737.—Fairly recent thrombosis in a mesenteric artery. Normal erythrocytes (stained yellow) are seen at top right. Erythrocytes which have lost some of their hæmoglobin are entangled among strands of fibrin (stained red) in the left half of the arterial lumen. There is no fibrinoid necrosis of the media. The stretched and attenuated internal elastic lamina can be seen as a fine black line in the left half of the arterial wall (Ektachrome. Hyaluronidase, picro-Mallory). (× 75.)

the appearances suggested infarction. On the antimesenteric border of the bowel were several dark-red patches varying in diameter from 0·5 cm. to 2 cm., over which the serous coat had lost its lustre. The related mesentery was œdematous but there was no evidence, either visible or palpable, of arterial or venous thrombosis. Enlarged firm lymph-nodes were present in the mesentery. The affected segment of ileum was resected and end-to-end anastomosis performed.

POST-OPERATIVE COURSE.—The child made an uneventful recovery. The bowels moved on the fourth day. He was returned to the Thoracic Unit at Mearnskirk Hospital on the thirteenth day with the wound soundly healed. He was allowed home twenty-seven days after laparotomy.

At a recent follow-up (Feb. 2, 1957) the boy appeared fit and well, though his mother stated that he had had several episodes of fairly severe abdominal pain since leaving hospital. Clinical examination was negative and a barium meal revealed neither delay nor obstruction in the small bowel. He went back to school on Feb. 20, 1957.

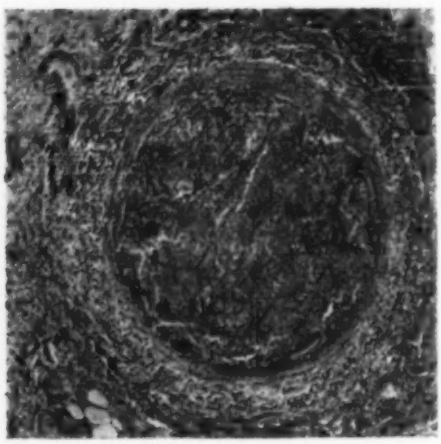

FIG. 738.—Old thrombosis in a mesenteric artery. Fibrin (stained red) occurs as broad bands round the circumference of the vessel in contact with the intima and running across the lumen. There is no fibrinoid necrosis of the media (Ektachrome. Hyaluronidase, picro-Mallory). (× 75.)

PATHOLOGICAL REPORT.—

Naked-eye Appearance of Specimen.—The specimen consisted of a portion of small intestine 350 mm. long with a fringe of 5 mm. of attached mesentery. The greater part of the peritoneal surface was congested and dark purple in colour. The intestine in this dark area had lost

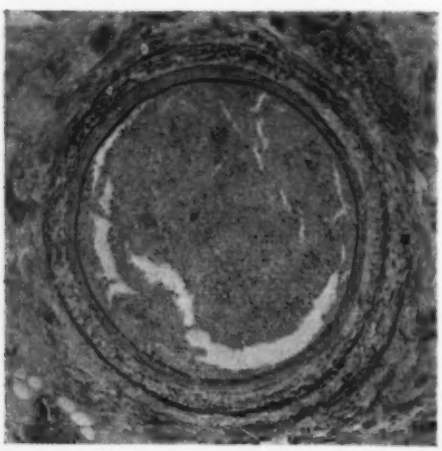

FIG. 739.—Distended mesenteric artery showing stretched and thin internal elastic lamina (stained black) with fragmentation of the fibres in the lower half (Ektachrome. Hyaluronidase, Weigert's elastic tissue stain and van Gieson). (× 75.)

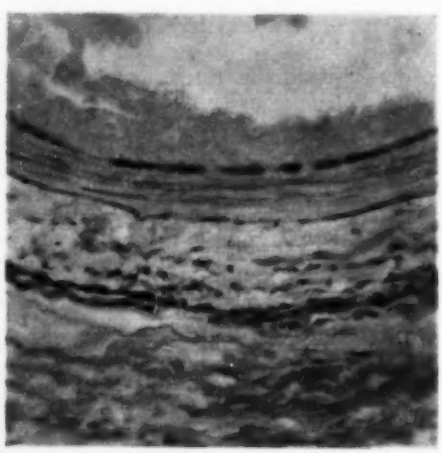

FIG. 740.—High power view of part of the arterial wall shown in Fig. 739. The fragmentation and rupture of the internal elastic lamina (stained black) are clearly seen together with attenuation of elastic fibres among the muscle of the media (stained red) (Ektachrome. Hyaluronidase, Weigert's elastic tissue stain and van Gieson). (× 348.)

its normal lustre and had a granular appearance but no actual necrosis was present (*Fig.* 736). The mucosa showed many small patches of superficial ulceration; these were most numerous towards the antimesenteric border.

Histological Examination.—The microscopical appearances are illustrated in *Figs.* 737–741 and are those of thrombosis of many of the small mesenteric arteries and arterioles, with the production of infarction of the bowel. Both recent and old thromboses are present. Recent thrombosis is shown by congestion of the arteries with some of the contained erythrocytes showing loss of hæmoglobin (*Fig.* 737). In other vessels in which the process is not so recent, a fibrin network is present with blood-corpuscles in the interstices. Still older thrombosis is shown by fibrin in the form of fairly broad annular bands in contact with the intima and as solid masses within the lumen (*Fig.* 738). In a few arteries organization of the clot is beginning to take place, the thrombus being replaced by cellular fibrous tissue in which capillary blood-vessels are beginning to appear.

There is an inflammatory cellular exudate of moderate degree in the media and adventitia of the thrombosed vessels (*Fig.* 741), but there is neither degeneration nor necrosis of the muscle of the medial coat. Inflammatory cells and erythrocytes occur throughout all coats of the intestine and they are especially numerous close to the surface in those areas which are denuded of mucosa.

Staining methods for demonstrating elastic fibres reveal that the internal elastic lamina of the arterial walls is stretched and fragmented (*Figs.* 739, 740). Instead of appearing as a well-defined continuous line, it is frayed and broken up into short lengths. The natural elasticity has been lost and the lengths of the elastic fibres are straight instead of showing regular undulations as a result of the inherent contractability. (For comparison, *Figs.* 742 and 743 show sections of a vessel in the wall of the ileum from a boy of the same age as the patient. A loop of ileum which had become kinked and was causing obstruction had to be resected. The vessels show congestion but the internal elastic lamina is intact and appears as a wavy but continuous line around the entire circumference of the arterial wall.)

PREVIOUSLY PUBLISHED CASES

Sealy (1953) was the first to report a post-operative vascular complication remote from the actual operation site. Following a classical operation for coarctation, his patient, a male of 22 years, developed severe abdominal pain on the third post-operative day. At laparotomy ten days after the onset of abdominal symptoms extensive infarction of jejunum and ileum was found. The patient died while resection of the affected bowel was being attempted.

Lober and Lillehei (1954) described 2 cases of 'necrotizing panarteritis'. The first was that of a boy of 12 years who complained of abdominal pain on the fourth day after resection of coarctation of the aorta. At laparotomy massive hæmoperitoneum due to rupture of an aneurysm of the splenic artery was found together with infarction of about three-quarters of the small bowel. The affected portion of ileum, 300 cm. long, was resected and end-to-end anastomosis performed. The patient died eight days later. The second case was a 46-year-old male who developed abdominal pain on the eighth post-operative day. At laparotomy on the tenth post-operative day 38 cm. of gangrenous jejunum were successfully resected.

Perez-Alvarez and Oudkerk (1955) reported a case of 'necrotizing arteritis' of the abdominal organs following operation for coarctation of the aorta. A 5-year-old boy complained of abdominal pain eight days after resection of the coarctation. At laparotomy on the eleventh post-operative day no abnormality other than mesenteric adenitis was noted. Following operation his condition continued to deteriorate and at a second abdominal exploration nine days later, about half of the ileum was found to be gangrenous; a portion 35 cm. long was resected and end-to-end anastomosis performed. Death supervened seven days after resection of the involved bowel.

Sealy reported another patient with infarction of the bowel after resection of the coarctation; in this case the gangrenous portion of ileum was successfully resected (Benson and Sealy, 1956). The patient was a boy aged 4 years who developed vague abdominal pain on the third post-operative day but did not develop signs of localized peritonitis until the sixteenth post-operative day. At laparotomy on that day 70 cm. of gangrenous ileum was resected and end-to-end anastomosis was performed. The patient made an uneventful recovery.

Ring and Lewis (1956), in a survey of abdominal pain following surgical correction of coarctation of the aorta, reported 4 patients in whom laparotomy was performed. Two were those previously described by Lober and Lillehei (1954). In the third mesenteric adenitis was found; the abdomen was closed and the patient made an uneventful recovery. The fourth patient had peritonitis and patchy necrosis of ileum and distal jejunum; 38 cm. of affected small intestine were resected and recovery was satisfactory.

Histological examination of the affected tissues showed that the essential lesion common to all reported cases was a fibrinoid necrosis of the arterial wall associated with an inflammatory cellular reaction. The smooth muscle-fibres of the medial coat were replaced to a varying extent by eosinophilic fibrinoid material. In many vessels the whole thickness of the muscle coat was affected along part of the circumference of the vessel, and the remaining muscle-fibres showed varying degrees of fraying, disintegration and degeneration; aneurysmal dilatation sometimes occurred as a result of stretching of the weakened parts of the vessel wall and in these dilated segments the arterial wall seemed to be composed only of adventitial fibrous tissue. In some vessels the muscle coats showed fibrinoid necrosis throughout the whole of their circumference and in these the intimal coat, too, often contained fibrinoid deposits. Minimal lesions involved only the inner part of the media. A profuse acute inflammatory cellular exudate usually occurred in the medial and adventitial coats of the affected vessels. Multiple, small thrombi (both recent and old) were noted in sections from material obtained at necropsy in the 3 patients who died after abdominal exploration. Lober and Lillehei (1954) stated that the internal elastic lamina was often preserved until the medial coat was almost destroyed. Benson and Sealy (1956), on the other hand, noted that the internal elastic lamina in the larger arteries was often fragmented, but no photomicrographs, stained to show elastic fibres, appear in either of these papers.

In the 3 patients who survived resection of necrotic intestine the vessels affected were the smaller arteries and arterioles in the mesentery and the bowel wall. In the 3 cases in which necropsy was performed

Table I.—CLINICAL AND PATHOLOGICAL FEATURES OF PATIENTS WHO REQUIRED LAPAROTOMY IN THE POST-OPERATIVE PERIOD FOLLOWING RESECTION OF A COARCTATION

AUTHORS	SEX AND AGE	NUMBER OF DAYS BETWEEN RESECTION OF COARCTATION AND			OPERATIVE PROCEDURE	RESULT
		Onset of Abdominal Symptoms	Onset of Localizing Abdominal Signs	Laparotomy; with Findings		
Lober and Lillehei (1954) *Case 1*	M 12	4 Epigastric pain	15 Abdominal distension. Patient became unconscious	15 Hæmoperitoneum due to ruptured aneurysm of splenic artery. Necrosis of three-quarters small intestine	Splenic artery ligated; 300 cm. ileum resected	Died 8 days after laparotomy. Autopsy: numerous thrombosed intercostal arteries below level of coarctation; numerous thrombosed mesenteric arteries; anastomosis of ileum healing
Lober and Lillehei (1954) *Case 2*	M 46	8 Pyrexia and vomiting	10 Abdominal distension, tenderness, and rigidity	10 Patches of gangrene in ileum and distal jejunum; mesenteric vessels appeared normal	Resection of 38 cm. of affected ileum	Recovery
Perez-Alvarez and Oudkerk (1955)	M 5	8 Generalized abdominal pain followed by localized pain round umbilicus	11 Laparotomy performed because of continued unremitting central abdominal pain	11 Mesenteric adenitis	Abdomen closed after inspection	Abdominal pain persisted. Melæna 5 days after laparotomy. Abdomen reopened 9 days after laparotomy; half of ileum necrotic; 35 cm. resected. Died 16 days after second laparotomy. Autopsy: acute arteriolar necrosis in intestines, urinary bladder, kidneys, and renal pelves; recent infarcts in kidneys and in left lobe of liver
Benson and Sealy (1956) *Case 1* (briefly reported by Sealy, 1953)	M 22	3 Pain in L. lower abdominal quadrant. On ninth day abdominal distension, vomiting, and ileus	13 Localized tenderness	13 Infarction of most of small intestine and proximal colon	Resection of affected gut attempted	Died during operation. Autopsy: liver showed large recent infarcts and paracentral necrosis; recent infarcts also present in kidneys, intestines and adrenals; interstitial hæmorrhages in gastric wall, pancreas, and renal pelves
Benson and Sealy (1956) *Case 2*	M 4	3 Vague abdominal pain. On ninth day slight abdominal distension	16 Severe abdominal pain in R. lower quadrant	16 70 cm. of terminal ileum gangrenous	Resection of affected portion of ileum	Recovery; discharged from hospital 12 days after laparotomy
Ring and Lewis (1956) J.S. (VH 850534)	M 3	7 Generalized abdominal pain, rigidity, tenderness; pyrexia, leucocytosis	9 Symptoms and signs more severe	9 Mesenteric adenitis	No operative procedure carried out. Abdomen closed after examination of peritoneal cavity	Recovery; discharged from hospital 18 days after laparotomy
Ring and Lewis (1956) EH (Associated Hospital)	M 46	8 Fever and vomiting	10 Abdominal distension, tenderness, and rigidity	10 Purulent peritoneal exudate; gangrenous patches along ileum and distal jejunum; mesenteric vessels not abnormal	Resection of 38 cm. of affected small intestine. Mesenteric arteries and arterioles showed medial necrosis with fibrinoid deposits and periarterial inflammatory cellular exudate	Recovery was uneventful
Present case	M 8	11 Lower abdominal colic, vomiting	14 Extreme tenderness and muscle guarding in L. iliac fossa. Rebound tenderness. Neutrophil leucocytosis. No fever	14 Purulent peritoneal exudate; several patches of necrosis in a segment of 30 cm. of terminal ileum; mesenteric vessels pulsating; mesentery thickened	Resection of 36 cm. of affected small intestine. Thrombosis of terminal arteries with NO medial necrosis but with disruption of internal elastic lamina	Recovery. Discharged from hospital 27 days after laparotomy. Several attacks of fairly severe abdominal colic during subsequent 6 weeks

the changes were diffusely distributed in the arteries of muscles and viscera supplied by vessels which arose distal to the site of the coarctation. In Lober and Lillehei's Case 1, with the ruptured aneurysm of the splenic artery, the breach in the vessel wall had occurred in an area of necrosis. In no case was any pathological change noted in the arteries arising proximal to the coarctation.

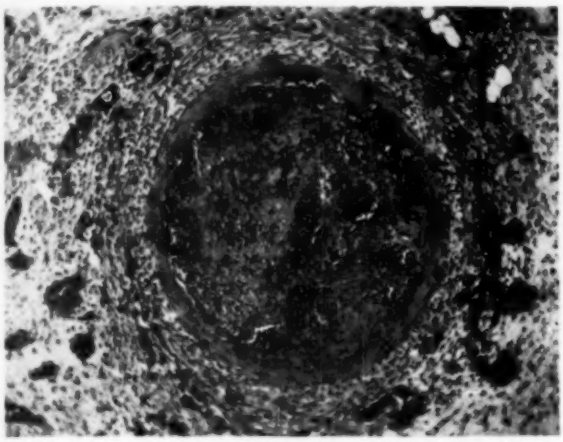

FIG. 741.—Established thrombosis of a mesenteric artery with a mild to moderate inflammatory cellular exudate in the adventitia. (Panchro line. No filter. Hæmotoxylin and eosin.) (× 75.)

The relevant clinical and pathological features in 8 patients in whom laparotomy has been carried out are summarized in *Table I*. All were male and were aged between 3 and 46 years. In each of the 3 patients who died, the length of necrotic intestine was over 300 cm.—much more extensive than in those who recovered. The length of small intestine resected in the 4 patients who survived this procedure varied from 36–70 cm. The interval between the onset of abdominal symptoms and laparotomy varied between 3 and 11 days in those who died, and between 2 and 13 days in those who recovered. One patient who recovered uneventfully was found to have only adenitis; there was no obvious intestinal lesion and the abdomen was closed after thorough inspection of the peritoneal cavity (Ring and Lewis, 1956). A similar case is mentioned by Cleland, Counihan, Goodwin, and Steiner (1956). It must be remembered, however, that mesenteric adenitis has been followed by extensive infarction of the ileum in one instance (Perez-Alvarez and Oudkerk, 1955). This means that all patients who develop clinical features which suggest an intraperitoneal lesion must be closely observed and that an exploratory laparotomy must be carefully timed.

DISCUSSION

In their article on arterial necrosis following operative treatment for coarctation of the aorta, Benson and Sealy (1956) discuss the possible aetiological factors. They conclude that the arterial necrosis is due to changes in the circulatory dynamics following the resection of the coarctation, the most important factor being the sudden sustained increase in the systolic pressure in the vascular tree distal to the site of coarctation. These writers bring forward much experimental evidence to support their contention.

In the case reported by us, the changes in the walls of the arteries in the resected bowel were also due to the effect of sudden hypertension in vessels unused to such a high systolic blood-pressure, but the microscopic appearances were significantly different from those noted by Lober and Lillehei

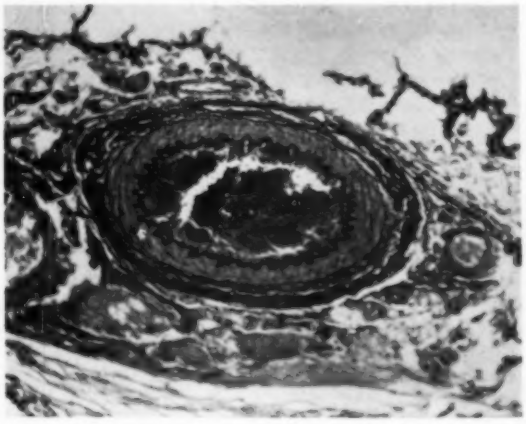

FIG. 742.—Normal appearance of the internal elastic lamina in a mesenteric artery for comparison with *Fig.* 739. In this photomicrograph the internal elastic lamina appears as a wavy but continuous line around the entire circumference of the vessel wall. (P 300. Wratten 110 minus blue filter. Hyaluronidase, Weigert's elastic tissue stain and van Gieson.) (× 75.)

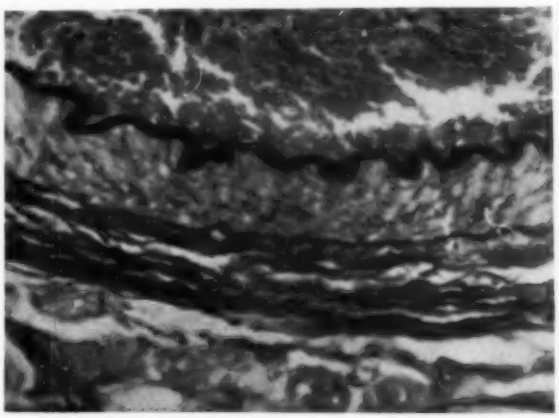

FIG. 743.—High power view of part of the arterial wall shown in *Fig.* 742. (P 300. Wratten 110 minus blue filter. Hyaluronidase, Weigert's elastic tissue stain and van Gieson.) (× 300.)

(1954), Perez-Alvarez and Oudkerk (1955), Benson and Sealy (1956), and Ring and Lewis (1956). All these authors reported that the most conspicuous histological features were fibrinoid necrosis affecting chiefly the muscle of the medial coat and an accompanying acute inflammatory cellular exudate ('necrotizing arteritis'); thromboses of varying age were seen in many of the smaller vessels.

In our case fibrinoid necrosis of the arterial wall is not seen and a periarterial inflammatory cellular exudate is only slight as a rule. The most striking microscopical feature is thrombosis in the smaller arteries and arterioles in the bowel wall. The age of the thrombus varies in different vessels, being

very recent in some and showing commencing organization in others. The internal elastic lamina is so severely stretched as to be broken up into short fragments. The resultant damage to the intimal cells is considered to be the direct cause of the thrombosis which evidently started at or soon after the resection of the coarctation and then extended over the ensuing 14 days.

The effects on the arterial blood-vessels of a sudden increase in the systolic blood-pressure have been studied by many investigators, using material from cases of malignant hypertension in man and from animals in which hypertension was produced experimentally. By partially occluding one renal artery in the rat, Wilson and Byrom (1939) were able to produce sustained hypertension with acute arteriolar fibrinoid necrosis in the vessels of the opposite kidney similar to the lesions seen in malignant hypertension in human patients. Their *Figs.* 740 and 741 illustrate considerable cellular thickening of the intima in a medium-sized artery, with marked stretching and fraying of the internal elastic lamina. Acute arterial lesions similar to those in the kidney in which the renal artery was not constricted also occurred in the pancreas, intestine, stomach, and heart, and occasionally in the adrenals, liver, and testis. The most severe lesions were in the terminal branches of the mesenteric arteries. This distribution of acute fibrinoid necrosis of arteries is similar to that found in malignant hypertension. The rise of blood-pressure in the experimental animals sometimes occurred as soon as 18 hours after constricting the renal artery. Even this short time is not comparable to the abrupt systolic hypertension which occurs in the arteries arising distal to the site of coarctation when the resecton and anastomosis have been completed and the clamps removed.

Wilson and Pickering (1938) showed that when hypertension is produced in rabbits either by constriction of both renal arteries or by constriction of one renal artery with removal of the opposite kidney, acute fibrinoid necrosis of the walls of arteries could occur. These lesions, which were identical with those of malignant hypertension, could be demonstrated in numerous organs but particularly in the intestines.

In the case reported here it is suggested that the pathogenesis of the infarction of terminal ileum was as follows: When the aortic clamps were released, there was a sudden marked increase in the systolic pressure in the arteries originating below the level of the coarctation. These arteries had been accustomed to a very low systolic pressure and the sudden increase in tension would cause stretching and rupture of the internal elastic lamina. Overdistension would be particularly liable to occur in the mesenteric arteries which are not supported by voluntary muscle and which are almost in line with the aorta itself. Stretching and fragmentation of the internal elastic lamina would cause damage to the intimal coat, providing a starting point for thrombosis; once thrombus has formed it is likely to spread. Anastomoses are poor between the terminal branches of the mesenteric arteries and when several adjacent branches become thrombosed, devitalization and then necrosis of the affected portion of the intestine will occur. The oldest thrombus in our case probably dates from the operation of resection. This was clinically silent for 10 days, although thrombosis was extending during that time. By the fourteenth post-operative day the bowel was permeable to intestinal contents and localized peritonitis was present and was producing clinical signs.

The present case showed no fibrinoid necrosis of the medial muscle coats and in this respect it differed markedly from the other six recorded cases. It seems likely that the abrupt hypertension in the mesenteric arteries after release of the aortic clamps was momentary and analogous to the experiments of Byrom and Dodson (1948), who forcibly injected fairly large quantities of Ringer's solution into the common carotid artery of the rat. They found that the systolic pressure at the lower end of the aorta rose immediately by 80–90 mm. Hg, but fell to normal in 2–3 sec. This procedure caused neither fibrinoid necrosis nor other lesions of the mesenteric arteries, but the blood-pressure before the injection experiments was within normal limits, in contrast to the very subnormal pressure in arteries distal to a coarctation. The onset of abdominal features in our patient was on the eleventh day after resection of the coarctation compared with an interval of 3–8 days in other recorded cases, so the process may have been slower with a consequently better chance of the anastomoses being effectively established. Evidence for the presence of continuing thrombosis is provided by the patient giving a history of several attacks of colicky abdominal pain after leaving hospital. The reason for the absence of fibrinoid necrosis of the muscle coats in the arteries of the present case and for its presence in the other reported cases is thus not clear. Possibly the rise in the systolic pressure in the distal aorta after removal of the clamps may have been more gradual because the operation was performed using hypothermia.

Although the authors who reported these other 6 cases considered that the sudden rise of arterial blood-pressure distal to the site of the aortic anastomosis was probably the most important aetiological factor in the production of the changes in arteries arising from the aorta distal to the coarctation, all were impressed by the resemblance of the lesions to those of periarteritis nodosa. Indeed Ring and Lewis (1956) suggested that the changes in two of their cases might be "examples of periarteritis nodosa in a limited portion of systemic circulation", although muscle biopsy in the lower limbs of patients who had abdominal symptoms but did not require laparotomy was negative. These authors, as well as Lober and Lillehei (1954) and Benson and Sealy (1956), listed the various drugs which their patients received in the period before and after resection of the coarctation, but concluded that none was a recognized allergen which might cause periarteritis nodosa. In our patient there is no suspicion of polyarteritis nodosa; there is no fibrinoid arterial necrosis, no aneurysmal dilatation, and there is only a mild periarterial inflammatory cellular exudate which is secondary to the intra-arterial thrombus.

Our case contributes a convincing and simple explanation of the aetiology. The most striking abnormality is the thin, overstretched, ruptured internal elastic lamina. This has been produced by the sudden expansile pressure of a greatly augmented

blood-flow which was pumped suddenly into vessels unaccustomed to such a high tension. This abrupt alteration in hæmodynamics is sufficient to explain the subsequent thrombosis in the mesenteric arteries and all the other pathological changes in our patient. There is no need to invoke, in addition, the presence of an allergen (known or unknown) to account for any of the microscopical or gross changes.

Why then do only a very small proportion of the patients apparently suffer in this way? When the aortic clamps are removed the greatly augmented arterial flow hurls itself into the distal hypoplastic vascular tree. The superior mesenteric artery and its branches are probably especially vulnerable to as in the case described in this paper, a localized infarction may result and require operation.

Such a hypothesis would explain the abdominal pain reported by several authors. In an article describing their experience of resection of the coarctation in 40 patients, Cleland and others (1956) reported that in the post-operative period 5 patients developed abdominal pain which was severe enough to cause concern but did not require laparotomy. Price Thomas, in a personal communication to Cleland, had a similar experience and in 1 patient a laparotomy was performed but no abnormality was found apart from mesenteric lymphadenopathy and congestion of the mesenteric vessels. Ring and

Table II.—INCIDENCE OF ABDOMINAL SYMPTOMS NOT REQUIRING LAPAROTOMY IN THE POST-OPERATIVE PERIOD IN PATIENTS WHO HAD A RESECTION OF A COARCTATION

AUTHORS	NUMBER OF PATIENTS IN SERIES IN WHOM RESECTION OF COARCTATION WAS CARRIED OUT	NUMBER OF PATIENTS WHO HAD ABDOMINAL SYMPTOMS NOT REQUIRING LAPAROTOMY FOLLOWING RESECTION	REMARKS
Clagett and Jampolis (1951)	62	1	Female experienced intense, boring, epigastric pain on ninth, tenth, and twelfth days after resection
Tonelli, Baisie, and Malizia (1954)	4	1	Female experienced "several anginoid crises"
Cleland, Counihan, Goodwin and Steiner (1956)	40	5	—
Ring and Lewis (1956)	73	14	Included 1 female. (Other 4 patients required laparotomy; *see Table I* and text)
Thoracic Surgical Unit, Mearnskirk Hospital	15	0	(The patient reported in this paper, who required laparotomy, was the only one in the series who had an abdominal complication)
Total	194	21 (10·8 per cent)	18 males; 3 females

this raised blood-pressure, partly because the widespread branching into the intestinal arteries and arcades leads to a rapid diminution in calibre of the individual vessels, and also because of the lack of supporting tissue in the mesentery.

The immediate distension of the smaller arteries and arterioles is probably followed by some degree of reactionary vasospasm. Thus, in the experiments of Byrom and Dodson (1948) in which repeated and forceful intracarotid injections of Ringer's fluid were used to cause transient hypertension, momentary blanching of small, irregular areas in the cortices of the kidneys occurred; this was ascribed to transient spasm of the capsular and cortical arteries. The reactionary vasospasm will protect the smaller vessels from further damage and will persist until minor intimal damage is repaired. Meanwhile there will be some ischæmia of the bowel presenting clinically as colicky abdominal pain. If the sudden overdistension of the arterioles results in a greater measure of intimal damage, then thrombosis will occur, and, if this is extensive, massive infarction of viscera will follow. Patients showing such changes are likely to be beyond surgical aid. Patients with less severe abdominal symptoms will probably recover spontaneously after a few days of discomfort or pain, but Lewis (1956) reported a series of 73 cases of coarctation of the aorta. There were 18 patients who had abdominal symptoms requiring treatment in the period immediately following resection of the coarctation. Fourteen had abdominal pain with vomiting, ileus, pyrexia, and leucocytosis. The onset of symptoms was between the first and tenth post-operative day. Treatment by gastric suction and intravenous fluids was followed by subsidence of symptoms within ten days. The remaining 4 patients who required abdominal exploration have already been mentioned. Clagett and Jampolis (1951) reviewed 70 cases in which thoracotomy was performed. The coarctation was resected in 62 patients and 1 female patient complained of intense epigastric pain on the ninth, tenth, and twelfth days after operation; but all investigations were negative. Tonelli, Baisie and Malizia (1954) described 1 female patient in whom the "post-operative clinical course [was] complicated by several anginoid crises", but no other details are given. The incidence of abdominal symptoms not severe enough to need laparotomy is shown in *Table II*. The four series quoted appear to comprise all those in the literature in which such symptoms are specifically mentioned. The number of cases varies considerably between the different

series, being low in that reported from the Mayo Clinic (Clagett and Jampolis, 1951) and high in that of Ring and Lewis (1956). The overall incidence is just over 10 per cent. The incidence of abdominal complications requiring laparotomy and operative treatment is very much lower; only 3 female patients experienced abdominal symptoms and all recovered without operation.

In a recent leading article in *The Lancet* (1956) on this problem of abdominal vascular complications following resection of a coarctation of the aorta, two questions were posed. First, what can be done to prevent the complications? With this in mind one might consider, on the one hand, the advisability of releasing the aortic clamps gradually, or again the use of hypotensive drugs. Secondly, what possible late effects might result from arterial damage to kidneys and other viscera? Only a follow-up of the patients over the years will provide an answer.

Finally, from a practical viewpoint one must be aware of the occurrence of such vascular changes and of their possible sequelæ. Whenever abdominal pain occurs post-operatively, laparotomy should be delayed until there is clear evidence that a serious and localized intraperitoneal lesion has become established; premature exploration may fail to reveal any significant abnormality but the patient may die later from the effects of a progressive infarction.

SUMMARY

A case of infarction of a portion of the terminal ileum following resection of a coarctation of the aorta is described, and the clinical and pathological features of 6 similar cases which have been reported are reviewed.

This complication arises from damage to the walls of arteries arising below the site of the coarctation. It is due to the sudden and sustained increase in the systolic blood-pressure which occurs immediately after the anastomosis of the aorta has been completed and the clamps have been released.

The most conspicuous histological features in the infarcted ileum in the 6 cases which have already been reported were: (1) Fibrinoid necrosis affecting chiefly the muscle of the medial coat of the arteries in the resected bowel, and (2) An accompanying acute inflammatory cellular exudate in all coats of the arteries ('necrotizing arteritis'). In the present case fibrinoid necrosis of the intestinal arteries was not seen and a periarterial inflammatory cellular exudate was slight; the most striking abnormality was the thin, overstretched, ruptured internal elastic lamina which was caused by the abrupt expansile pressure of a greatly increased blood-flow pumped suddenly into poorly supported vessels unaccustomed to such high tension. The resulting damage to the intimal cells caused thrombosis which slowly extended until the wall of the ileum was devitalized.

Abdominal pain which does not require laparotomy occurs after resection of the coarctation of aorta in about 10 per cent of cases. This pain, which is usually colicky, is probably due to ischæmia of a localized segment of bowel caused by damage to the walls of the intestinal arteries, with or without vasospasm.

ADDENDUM

Since this article was submitted for publication, Hurt and Hanbury (1957) have reported the clinical and pathological findings in a boy, aged 10 years, who developed abdominal distension and colic on the third day after resection of a coarctation and signs of intraperitoneal hæmorrhage on the seventeenth post-operative day. At laparotomy, 1½ pints of blood were found in the peritoneal cavity; it had come from a ruptured aneurysmal dilatation on a mesenteric blood-vessel; many such localized dilatations up to 6 mm. in diameter were present on the mesenteric arteries, and subperitoneal hæmorrhage extended into the adjacent wall of a segment of ileum causing narrowing of the lumen. The affected loop of ileum (250 mm. long) was resected and end-to-end anastomosis performed. Microscopical examination revealed no infarction of the bowel wall, but there was aneurysmal dilatation of the mesenteric arteries and an intense necrotizing arteritis with replacement of the media by fibrinoid; the submucous arteries in the bowel wall also showed necrotizing arteritis; in addition, fragmentation on the internal elastic lamina is clearly shown in their *Fig.* 5, and these appearances are similar to those seen in *Fig.* 739 of the present paper.

We are glad to express our gratitude to Mr. R. S. Barclay, Mearnskirk Hospital, and to Mr. Robert Mailer, Victoria Infirmary of Glasgow, for permission to publish. We are grateful to Dr. W. B. Davis and to Mr. Robert Mailer for much helpful advice and criticism during the preparation of this paper; to Dr. A. M. MacDonald, Pathology Department, Royal Hospital for Sick Children, Glasgow, for supplying the material from which *Figs.* 742 and 743 were prepared; and to Mr. H. C. Gray, A.R.P.S., for assistance with the photography. We also wish to thank the Board of Management of the Glasgow Victoria Hospitals for a generous grant for expenses.

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BASAL-CELL CARCINOMA ARISING IN A PARANASAL DERMOID CYST

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PARANASAL dermoids, situated along the line of junction of nose and cheek, are uncommon; also, though squamous-cell carcinoma has been known to develop in dermoid cysts, we are unaware of any record of basal-cell carcinoma arising therein.

The following case presents a combination of these unusual features and is, therefore thought to be worth recording.

CASE REPORT

A man, aged 52 years, noticed a swelling beside his nose at its junction with the right cheek. This increased until it reached the size of a walnut and then, after he had noticed it for about a year, it burst externally, discharging thick whitish material. A sinus persisted.

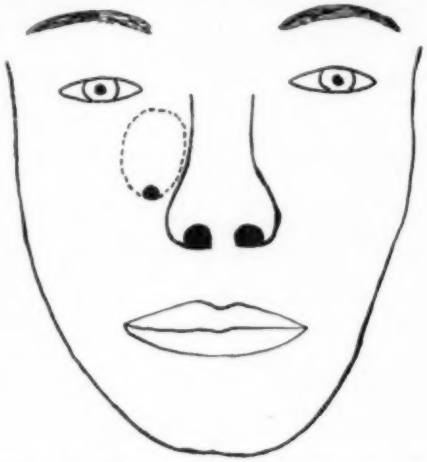

FIG. 744.—Drawing showing the position of the dermoid cyst.

After a further two years or so a surgeon, who was treating him for a fracture of the tibia and fibula, noticed the lesion, together with a suspicious nodularity of the margin of the sinus, and carried out a biopsy of this as a preliminary to excision of the cyst. The biopsy showed the presence of basal-cell carcinoma. As it was thought that wide excision was indicated, which would involve skin replacement, the patient was referred to one of us (J. N.) and admitted to the Regional Plastic Surgery Centre in November, 1956, about three years after the swelling was first noticed and two years after the sinus became established.

ON EXAMINATION.—There was an oval swelling about 3·5 cm. × 1·75 cm. situated in the concavity between the right side of the nose and the right cheek (*Fig.* 744). At its lower end, just above and lateral to the right ala nasi, was a sinus 3–4 mm. in diameter: a probe passed into this entered a cavity in the swelling. The skin over the swelling was slightly œdematous but not otherwise abnormal except for the presence of the sinus.

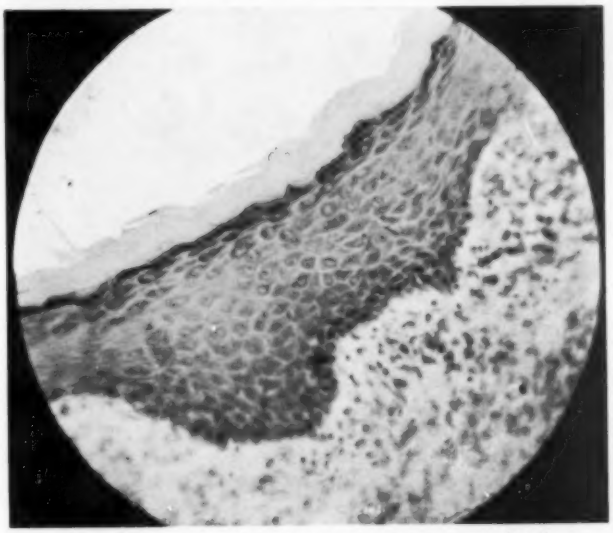

FIG. 745.—Part of cyst wall showing squamous epithelium lining. H. and E. (× 48.)

AT OPERATION.—The swelling was excised together with the whole of the skin overlying it and that surrounding the sinus. It comprised a cyst with a thick, firm wall, which at one point (near the ala nasi) was adherent to the lining of the vestibule, and which at its upper end tailed off into a solid fibrous cord attached to the fronto-nasal process in a manner reminiscent of dermoids seen elsewhere (e.g., nasal, 'external angular', or preauricular dermoids). Nothing about the appearances at operation

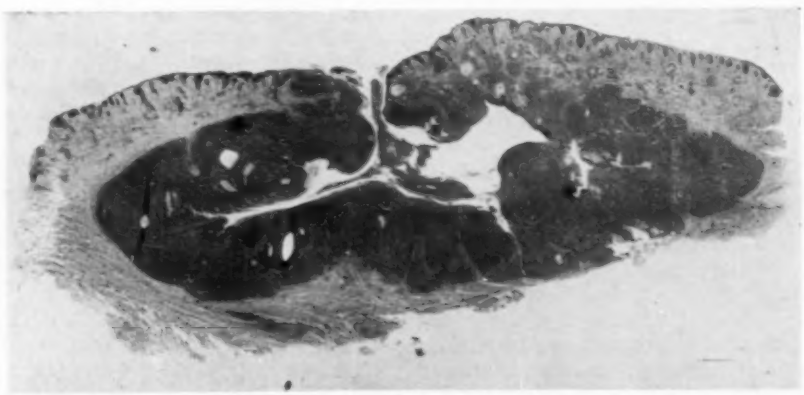

FIG. 746.—Low-power view showing the cyst surrounded by the tumour tissue. H. and E. (× 3·5.)

particularly suggested malignancy. The defect was closed by a transposed flap from the cheek.

PATHOLOGY.—The operation specimen consisted of an ellipse of skin which, after fixation, measured 3·2 cm. × 1·0 cm. with subcutaneous tissue to a depth of 1·0 cm. Towards one end of the skin was a small sinus, which on sectioning the specimen was seen to lead into a thick-walled cavity. The wall was composed of firm white tissue, and the cavity was empty.

Microscopically the cyst was lined by keratinizing squamous epithelium (*Fig.* 745), beneath which a more or less uniformly wide band of tumour tissue surrounded the cyst (*Fig.* 746). The tumour was composed of rounded masses of cells, the marginal parts of which showed a distinct layer of columnar cells; the central parts consisted of polyhedral and ovoid cells devoid of any special arrangement, and minute cysts were present in some of the epithelial masses. The appearances were those of a basal-cell carcinoma (*Fig.* 747).

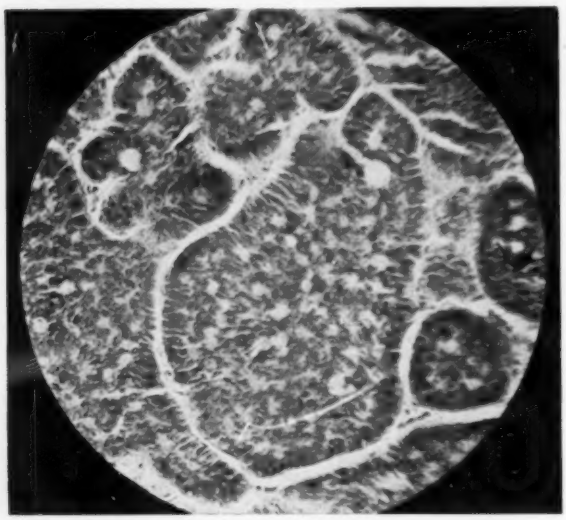

FIG. 747.—Part of the tumour showing the structure of a typical basal-cell carcinoma. H. and E. (× 192.)

The cyst had the characteristics of a dermoid cyst, and, in the absence of any previous injury to the face, which might have led to implantation, it must be regarded as a congenital sequestration dermoid, which could be expected to occur at this site.

DISCUSSION

1. Paranasal dermoids are apparently uncommon. According to the classical theory of facial development, advanced by Durscy (1869), His (1892), and others, and still quoted in most modern text-books of embryology (e.g., Arey, 1954), there is a line of fusion between the lateral nasal process and the maxillary process; and even if the alternative theory of the development of the middle third of the face, as put forward by Veau (1938) and others, is accepted, whereby a continuous ectodermal hood is supposed to become invaded by mesoderm, yet the formation of the nasolachrymal duct has still to be accounted for; and this is usually said to take place by the sequestration and subsequent canalization of a cord of ectoderm. By either theory, therefore, this region is a likely one for the formation of sequestration dermoids; and Forgue (1948) shows it as such in a diagram illustrating the sites of facial dermoids.

Nevertheless it is difficult to find references in the literature to dermoids in this situation. They may of course have been seen, but not reported for the very reason that they are theoretically probable and their apparent uncommonness has not been realized.

According to New and Erich (1938), 1495 dermoid cysts were seen at the Mayo Clinic during the period 1910–1935 inclusive. Of these 103 were situated in the head and neck; their situations are given, and none of them was paranasal, whereas the relatively common 'external angular' dermoids numbered 31. It is therefore reasonable to say that paranasal dermoids are at least uncommon.

2. That squamous-cell carcinoma may develop in sequestered squamous epithelium has been recognized for many years. This is implicit in the term 'branchiogenic carcinoma'. Willis (1948 a) has pointed out the difficulty of being sure, in the absence of a careful autopsy, that a squamous-cell carcinoma situated deeply in the neck is not secondary to a small hidden primary elsewhere, but he has seen two cases which in his opinion, and even by his high standards, probably originated in branchial remnants. In the series referred to above, New and Erich encountered one case in which a squamous epithelioma developed in a dermoid involving the soft palate.

We have not, however, found any previous record of basal-cell carcinoma developing in a dermoid cyst.

3. It is interesting that several authors have suggested a possible dysontogenetic origin for basal-cell carcinoma, and have pointed out that the sites of basal-cell carcinomata tend to be related to the supposed 'lines of facial fusion' while the sites of squamous-cell carcinomata do not (Glasunow, 1933; McFarland, Ciccone, and Gelehrter, 1935). Others have been unimpressed by this theory (e.g., Willis, 1948 b).

The present case is one at least in which basal-cell carcinoma did develop in what must be presumed to have been sequestered squamous epithelium. We mention this as a point of interest, but draw no conclusion from it.

SUMMARY

1. Paranasal dermoid cysts are uncommon, and basal-cell carcinoma arising in a dermoid cyst is apparently rare.

2. A case is described in which basal-cell carcinoma was found to have originated in a paranasal dermoid cyst.

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INTRA-HEPATIC ANATOMY

A SURGICAL EVALUATION

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DURING the past half century there has been an increasing awareness that the various duct structures of the liver are arranged in an orderly and segmental fashion, and that the surgeon who contemplates operations upon the liver should be familiar with this arrangement. In the main, two different methods have been used to investigate intrinsic hepatic anatomy; one involves the injection of radio-opaque dyes; the other the making of solid casts of the ducts. There can now be little argument about the broad outlines of the subject, but some questions of surgical importance receive conflicting replies from different investigators. It is the purpose of this article briefly to review the literature, to define the limitations of the usual methods of investigation, to describe some studies with these methods and also with a different technique, and to attempt an evaluation of the surgical importance of our knowledge of this subject.

FIG. 748.—Relation of portal vein to hepatic vein, seen dorsally (*after Glisson*, 1659).

THE LITERATURE

The first detailed account of intrahepatic anatomy was given by Glisson, 1659, in his comprehensive treatise *Anatomia Hepatis*. He included some beautiful and extraordinarily accurate illustrations of the relationship between the portal and the hepatic venous systems. Comparing *Fig.* 748, which is a reproduction of one of those illustrations, with the plate of a cast of those two systems (*Fig.* 749), and reflecting that Glisson's results were obtained without modern injection-corrosion techniques, one is struck anew by his tremendous capacity and application.

Rex (1888) and Cantlie (1898) gave the first descriptions of the true plane of subdivision of the liver into *right* and *left halves*. Hjortsjö (1951) carried these observations further by means of stereoscopic X-ray studies, particularly of the portal veins and bile-ducts. He described the commonest pattern of these structures, and recognized that the two liver halves could be further subdivided into smaller segments on the basis of the portal vein branches. Fainsinger (1950), like Hjortsjö, was principally concerned with the applications of hepatic structure to radiology, and he accordingly confined his observations to the bile-ducts. Another and most important contribution to the study of intrahepatic bile-ducts was the investigation by Healey and Schroy (1953) of a hundred livers. They were able to quote percentage frequencies for all the minor and major variations of pattern that they encountered. From 1952 onwards, Couinaud, in France, was publishing a series of articles on all the hollow systems and their possible variations. They represent an extraordinarily detailed corpus of anatomical fact—perhaps rather more detailed than the surgeon requires. However, Couinaud has also ably expounded (1954) a surgical interpretation of this mass of knowledge, and to his interpretation reference will later be made.

The most comprehensive recent reviews of the whole subject are given by Gans in 1955 in his monograph *An Introduction to Hepatic Surgery*, and by Couinaud (1957).

METHODS OF INVESTIGATION

1. The Injection of Radio-opaque Dyes.—The bile-ducts, being a closed system, are particularly suitable for study. The best medium as regards the quality of the resulting radiograph is 'viscous neo-hydriol', but once this material has been injected into a duct it is extremely difficult to wash out again. That duct, then, cannot be satisfactorily filled with plastic in order to prepare a cast.

FIG. 749.—Cast of the hepatic and the portal venous systems, seen dorsally. Hepatic veins pale, portal veins dark. The plastic cannulæ through which the veins have been filled have been left in situ.

The great disadvantage of the method is that it is not truly three-dimensional. Stereoscopic films have

been taken in all cases and produce a very striking impression: but even with the added precaution of a 45°-oblique film the radiographs can be difficult to interpret, and even definitely misleading.

The technical details have been as follows: 'Ilfex' film, 4½ sec. at 20 ma. and 40 kv.; film distance 36 in., transverse shift 4 cm.

2. Injection-corrosion Techniques.—A duct system is injected with a liquid which later hardens

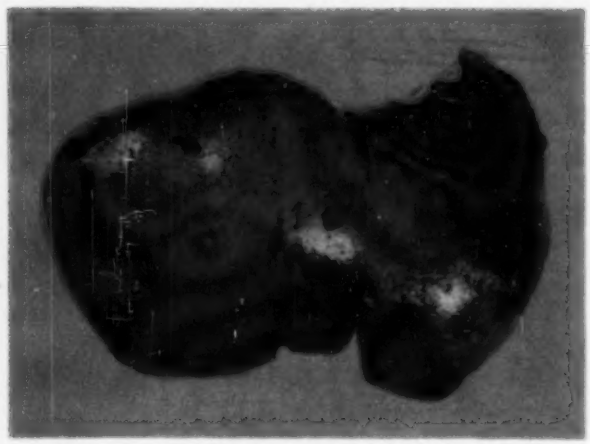

FIG. 750.—The right hepatic duct has been injected with a red dye, the left with a blue dye. The *principal plane* is thus demarcated on the surface.

to form a solid cast. The liver substance is then removed by acid digestion. Many materials are available for making such casts: the one used in this study is called 'Marco Resin SB 26C' (Scott Bader & Co., Ltd.). This substance is supplied with three other compounds—the 'monomer', the 'catalyst-H' powder, and the 'accelerator E'. Using the formula:

Resin SB 26C	100 g.
Monomer	40 g.
Catalyst H	4 g.
Accelerator E	4 ml.,

the second ingredient, and then the third, is mixed with the resin. At this stage a pigment may be included, and the mixture is stable at least for a few hours. However, when the accelerator has been added setting will occur in 15–20 min. at 20° C.

As regards the technique used, it has not been found necessary to employ many of the precautions detailed by Tompsett, working with lungs (1952) or with livers (1956). Good results will be obtained provided the hollow structures have been well washed out. This goal may be achieved by closing one end of the cut segment of inferior vena cava, with which the liver has been removed, and tying a cannula into the other end. The cannula is then attached to a running tap overnight. Afterwards, the duct systems being studied are also cannulated. The liquid plastic may be run in under gravity in the case of the portal and the hepatic venous systems, but requires to be injected by syringe into the smaller bile-ducts and arteries. Filling the liver under water is an aid to preserving the normal shape of the organ, and allows maintenance of a warm environmental temperature in a cold room.

The filled liver must be allowed to stand for twenty-four hours (at room temperature) to permit 'maturation' of the plastic. The capsule is scored in several places, and then the liver immersed in concentrated hydrochloric acid for a week. The macerated liver-tissue can now be washed off the cast by a jet of water through a Pasteur pipette, combined with judicious nudging with the end of the pipette.

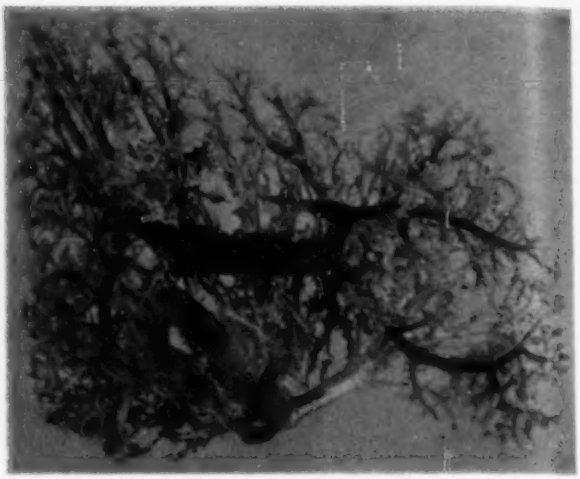

FIG. 751.—Cast of all four hollow systems: Colourless, Hepatic veins; Blue, Portal veins; Red, Hepatic arteries; Green, Bile-ducts. The liver has been turned upwards and is viewed from the front.

The cast method has the advantage of providing a durable and three-dimensional representation of the duct systems, but suffers from a number of drawbacks. The technique is time-consuming, and the finished product occupies much space. Furthermore, the accuracy of the procedure is difficult to control: washing a specimen may take 20–30 hours and it is a physical impossibility to avoid breaking off some of even the larger branches in the course of this task.

The most serious objection to the method, however, is that it involves the destruction of the liver surface. Therefore it cannot give with precision the very information that the surgeon most requires. If there be planes of relative bloodlessness which the surgeon might utilize in cutting the liver, he must know their exact location with reference to surface-markings on the viscus as it presents at operation. It is true that one can quite easily judge approximately the position of, say, the plane of the falciform ligament on a completed cast, but it is important to realize that such judgment is only approximate. Some of the statements in the literature about bloodless planes happen to be exactly true : but it will be shown later that some are not.

3. Injection of Coloured Dyes.—To meet the above difficulty, a leaf has been taken from the book of the workers on pulmonary morphology (Brock, 1946).

If a coloured dye such as methylene blue is injected along the hepatic duct of a well-washed liver, it permeates in a few minutes to the surface in a pattern of dots and patches that tend, but only to a limited extent, to coalesce with time. If the right and left hepatic ducts are separately isolated by dissection at the porta, and injected each with a

differently-coloured dye, a demarcation will appear on the surface of the liver between the areas drained by each duct (*Fig.* 750).

THE MATERIAL

This investigation has involved the study of 58 livers by all three methods previously described, though by no means all the methods have been used on every liver. It is possible to employ the coloured dye method, and follow it with either radiology or the plastic, but it has not been found possible to obtain a good cast after the ducts have been injected with neohydriol, or if a radio-opaque material is included in the resinous mixture. The livers were obtained during the course of routine autopsies. If the pathologist conducting the post-mortem was satisfied that it was not necessary to section the liver, he removed it from the cadaver with as long a length as possible of the duct structures in the free edge of the lesser omentum, and with a section of inferior vena cava on the back of the liver. Sometimes, more or less diaphragm was included in the specimen.

THE RESULTS

To the surgeon, the liver is an organ that bleeds excessively when cut; to the student of gross anatomy, the liver is preponderantly *hepatic veins*. *Fig.* 749 emphasizes the great bulk of these veins in comparison with the total volume of the organ. They drain into the inferior vena cava near the superior margin of the liver as three main stems, the *right*, the *middle*, and the *left* hepatic veins. To be exact, the middle usually joins the left vein before entering the cava. The hepatic veins run *upwards*, defining three corresponding segments of liver. Each segment drains more or less exclusively by its hepatic vein, and the extent of anastomosis between these segments is uncertain, though most investigators agree that this occurs (Elias and Petty, 1952; Gans, 1955). It will be seen from the cast illustrated that the left vein drains the 'classical' *left lobe*, that is, the area to the left of a sagittal plane through the liver attachment of the falciform ligament. This plane may be called, in brief, *the plane of the falciform ligament*.

There are some *lesser hepatic veins*, variable in number and exact arrangement, which drain into the front of the inferior vena cava at some distance below the origin of the three main veins. They pass backwards directly from the liver substance, and are of obvious surgical importance.

The portal veins occupy less than half the volume of the hepatic veins. 100 ml. of plastic was more than enough to fill the portal tree in all the livers studied, whereas 200 ml. was occasionally inadequate for the hepatic veins (*Figs.* 749, 751). At the porta

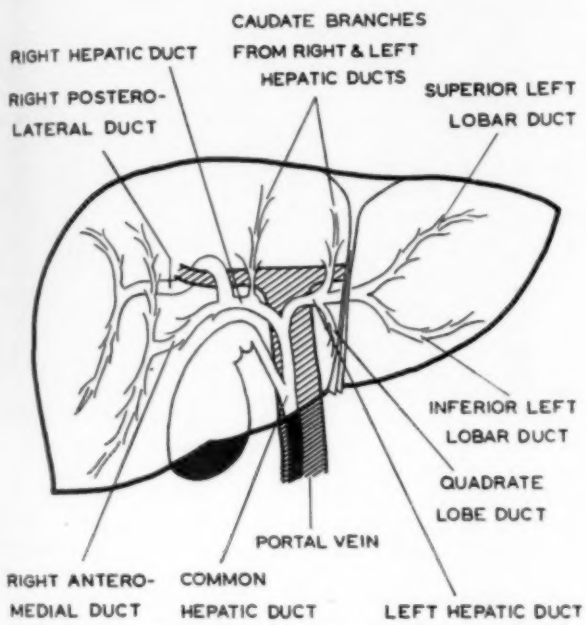

FIG. 752.—The commonest bile-duct pattern. The liver is viewed from the front and the common bile-duct, with the portal vein behind it, passes up behind the inferior edge of the liver.

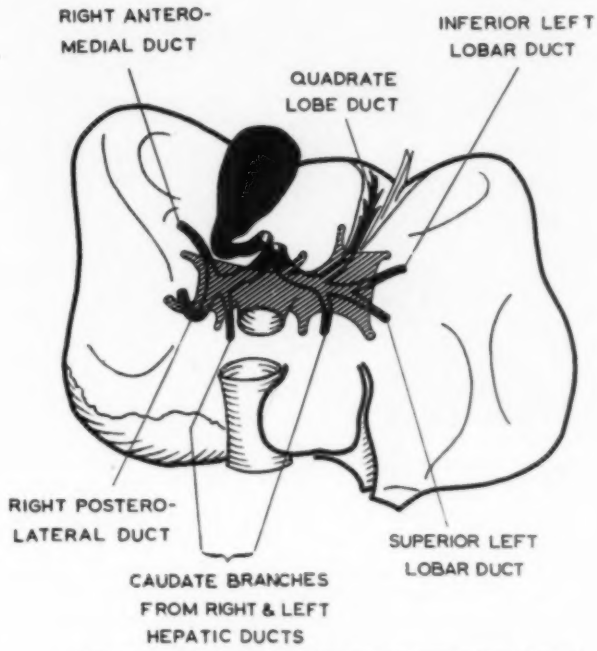

FIG. 753.—The liver has been turned upwards to show a common arrangement of structures in the portal fissure. Note the left division of the portal vein turning forwards to terminate as the blunt saccus, and the curve of the postero-lateral duct over the right division.

Key to *Figs.* 752, 753.

hepatis the main trunk of the portal vein divides into a *right* and a *left* division, the whole forming a T-junction. The right division is small and may even be non-existent, in which case branches to the right half of the liver take immediate origin from the portal vein trunk. The left division is much longer,

FIG. 754.—Coronal section through a liver of which the right hepatic duct has been injected with a dark dye. Note that the cut central hepatic vein (in which the point of the artery forceps lies) marks the central boundary of the injected area, i.e., the *principal plane*.

and proceeds transversely to the left along the porta hepatis, between quadrate lobe and caudate lobe, until it reaches the fissure for the ligamentum teres. This fossa lies in the plane of the falciform ligament. The division turns forwards along the fossa for about an inch, to end as a blind termination, usually called the *saccus*. In this part of its course it lies between quadrate lobe and left lobe.

From the portal vein, or from its divisions, branches to the liver substance take origin. Their general direction is *outwards*, and so in many parts of the liver the portal vein branches will be travelling at right angles to neighbouring hepatic vein branches. The two systems are entirely distinct.

The remaining pair of duct systems, the bile-ducts and the hepatic arteries, are both very much smaller than the portal veins. In terms of capacity for Marco Resin, the bile-ducts vary between 10 and 40 ml.; the arteries are not much larger. A cast in which all four hollow systems are injected (*Fig.* 751) shows that bile-passages and arteries closely cling to portal vein branches in their course through liver substance. Dissections in the region of the porta show that this association commences where the branches first plunge into the liver. At the other end of the scale the association is represented by the 'portal triad' familiar to the histologist. This intimate relationship of the three sets of vessels justifies dignifying the combination as a composite system for which the adjective 'Glissonian' has been suggested by Kiernan (1833). Information about the system can be gained by injecting any one of its three components.

The branches of the Glissonian system supply the liver in a segmental fashion: that is, a branch receives no anastomoses from neighbouring ones, and so supplies completely the sector of liver with which it is associated, and only that sector. No exception to this statement has been met in any of the casts made in this series, or as a result of radiological or coloured dye studies. Other observers have remarked on this point, e.g., Elias (1955), reviewing some earlier work of his own (Elias and Petty, 1952), and Gans (1955 b). However, Segall (1923) did describe some small and insignificant anastomoses between the right and left hepatic arteries.

The fact that bile-duct, hepatic artery, and portal vein originally divide into a right and a left branch implies that the liver must be interpretable on the basis of the Glissonian system, as two halves about a '*principal plane*'. This plane must be closely considered because of its importance in hemihepatectomy. *Fig.* 750 shows the anterior aspect of a liver of which the right hepatic duct has been injected with a red dye, the left with a blue. It will be seen that the principal plane is far to the right of the plane of the falciform ligament. It starts below at the apex of the gall-bladder notch and extends upwards to the point where the left hepatic vein debouches in the inferior vena cava. This specimen also showed, when viewed from behind, that the plane corresponds exactly to the groove that bisects the gall-bladder bed. Above the region of the porta there is no surface marking to indicate its position, but approximately it halves the caudate lobe. The demarcation is very sharp, but it is not everywhere very regular. Note, for example, that a large bulge of right half into left half is apparent in the upper part of the plane on the anterior surface of the liver.

FIG. 755.—The right posterolateral duct (arrowed) takes separate origin from what is apparently *left* hepatic duct.

In the depths of the liver it is important to recognize that the central hepatic vein lies in the principal plane (*Fig.* 754).

Study of the further subdivision of the liver necessitates familiarity with the main bile-duct branches. The commonest pattern of the biliary tree

is diagrammatically represented in *Fig.* 752. The common hepatic duct divides into *right* and *left hepatic ducts*: the former drains the right half of the liver by two main ducts, an *anteromedial* and a *posterolateral*, while the latter supplies a *branch*, or

FIG. 756.—A single duct draining the quadrate lobe is arrowed.

two branches, *to the quadrate lobe* and then, crossing the plane of the falciform ligament, divides into a *superior* and an *inferior* branch to the left lobe. Both right and left hepatic ducts severally originate a *branch* or *branches* to the caudate lobe, which it will

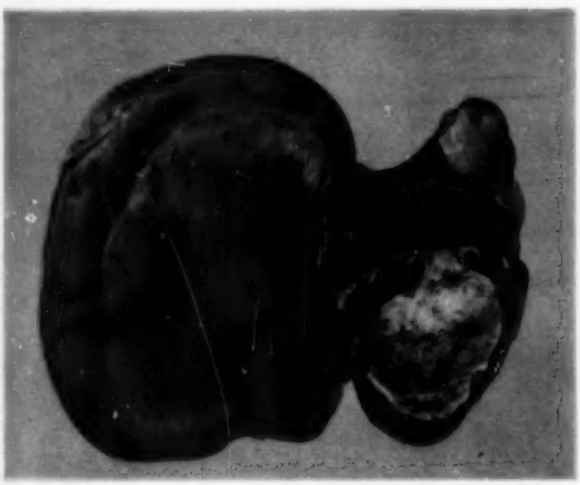

FIG. 757.—A quadrate lobe duct such as that shown in *Fig.* 756 has been injected with a blue dye; the duct to the anteromedial zone of the right lobe has been injected with a red dye.

be remembered is partly in the right half of the liver, partly in the left. Note how the right anteromedial duct curves over the right division of the portal vein.

Of vital importance to the surgeon is a knowledge of how much of the Glissonian system can be dissected at the region of the porta before it plunges into liver substance (*Figs.* 753, 761). The hepatic artery divides much earlier than the portal vein or the bile-duct, and its branches lie correspondingly more superficial. Their pattern of arrangement is very variable (Healey and Schroy, 1953; Couinaud, 1954), but by the time the deeper biliary and portal branches have been identified the operator can be in no doubt about which arterial branches correspond with the ducts and veins in which he is interested. The arteries are therefore, for simplicity, not shown in the diagram.

The common hepatic duct usually divides late, when it has almost come to lie between portal vein bifurcation behind, liver in front. Its branches in fact tend always to bear this inaccessible relationship to the portal system. This is particularly true of branches to the caudate lobe, which are never easily visualized, and also, to a less extent, of both the upper and the lower branch to the left lobe. These latter originate by bifurcation of the left duct, frequently at a point completely overlain by the left portal vein as it turns forwards as the saccus. However, the bifurcation is quite often proximal to the bend, and in such a case the two branches can be separately identified with greater ease.

It is true that the two branches to the left lobe can be picked up again as they emerge from cover of the saccus farther to the left. However, the biliary branches most easily located at the hilum are those to the quadrate lobe, and to the right half of the liver. The first subdivision of the right duct into anteromedial and posterolateral ducts can always be found, and quite often (Healey and Schroy quote a figure of 28 per cent) these second-order ducts in fact originate directly from the common hepatic duct (*Fig.* 755). More peripheral subdivisions than those just described cannot be dissected at the porta without removing liver tissue.

FIG. 758.—The anterior surface of the liver faces downwards. The right anteromedial duct has been injected with a blue dye, and the posterolateral with a red. *See* text for full description.

It is now possible to consider further segmentations in the right and left halves of the liver.

The first question that arises is whether the plane of the falciform ligament is also a plane of internal subdivision. Previous authors already mentioned have answered this question in the affirmative, as a result of studies with casts in which it was found

that a sheet of paper could be interposed between neighbouring branches along this plane. However, Healey and Schroy (1953) remarked that in 20 per cent of livers biliary branches may cross the plane in its lower third from right to left—that is, branches traverse the quadrate lobe and cross the fissure for the ligamentum teres to drain a small area of left lobe of liver (*Fig.* 755). Notwithstanding this exception, on the whole it proved true when dissecting hilar structures that the left lobe of the liver may be considered an independent entity as regards the Glissonian system. It is certainly true (and this is surgically the more important fact) that no element of this system originating to the left of the plane of the falciform ligament crosses the plane to supply an area to the right of the plane. It would appear justifiable, therefore, to retain the classical definition of *right* and *left lobes* separated by this plane.

In the right lobe we may consider first the duct or ducts to the quadrate lobe. Sometimes, this duct takes origin directly from the common hepatic duct, and is the only duct draining the quadrate lobe (*Fig.* 756). It was in such a case that blue dye was injected along the quadrate lobe duct, and the area marked out on the liver surface thereby was found to be not only the traditional quadrate lobe, on the back of the liver, but also the whole area between the principal plane and the plane of the falciform ligament on the front (*Fig.* 757). The caudate lobe is thus a structure superimposed on the upper half of the posterior surface of the *quadrate lobe*, as defined by the new concept.

We now come to the right half of the liver, supplied, as has been seen, by an anteromedial and a posterolateral duct, which may be considered to define corresponding *zones*. In a series of livers, one or both of these ducts has been injected with a coloured dye, thereby defining on the anterior aspect the line in which the plane between the two zones meets the surface. This line is extremely variable in position, and is not related to any obvious surface landmarks. Moreover, sagittal sections cut through a specimen in which the posterolateral duct had been injected with a red dye, the anteromedial with a blue, illustrate that though the distinction into posterolateral and anteromedial zones is sharp near the principal plane (*Fig.* 758), yet more to the periphery there is much overlapping. There is no transgression of the rule of segmentation, that is, no section shows mixing of the dyes. But a section near the periphery showed some red dots amidst areas preponderantly of blue dots, and vice versa. The effect is that the surgeon's blade would find this part of the 'plane' almost as well vascularized as any randomly selected area of liver.

Fig. 758 also shows another point of considerable negative importance. When Couinaud and Gans advocate operations involving transecting the liver along this plane they describe the right hepatic vein as running in the plane and therefore being a valuable guide to its location. This statement is only approximately true: the hepatic vein cut across in the section is obviously embedded entirely in the posterolateral lobe.

A final disadvantage of this 'plane' as regards surgery is that it is far from plane. Its contour defies description, especially owing to the variation in proportionate size of anteromedial and posterolateral zones. However, as an approximation one may describe the former as being a quarter of a sphere, one plane surface being the anterior surface of the liver while the other is the principal plane, and this lobule is ensheathed from behind and laterally, and also to a varying extent from above and below, by the posterolateral zone.

In view of the preceding considerations, the question of what to call this plane presents some difficulty. The terms employed by previous authors have always implied the endowment of properties which the plane does not possess, e.g., right interlobar fissure (Gans). It is therefore suggested that it be called the '*(right) indefinite plane*', a name that might remind the surgeon that its importance to him is secondary. Likewise, the two sub-units of the right half have been referred to by the vague term '*zones*', rather than by the more precise descriptions of 'lobes'.

SURGICAL APPLICATIONS

These will be considered under the following headings: bile-ducts in danger during surgical operations on neighbouring structures, resections of liver, local and segmental; and intra-hepatic cholangioenterostomy.

Bile-ducts in Danger.—Healey and Schroy (1953) emphasized the surgical importance of what they called the 'subvesical duct'. This duct usually arises from the duct to the anteromedial zone of the right lobe or from the right hepatic duct, and runs very superficially along the gall-bladder bed, more or less along the groove that marks the principal plane. It may even emerge from the liver and lie in the connective tissue between liver and gall-bladder. In any event, it is very liable to be opened during cholecystectomy, as the bladder is stripped off the liver. Healey describes an incidence of 34·5 per cent for a superficial duct of this nature. The present series is smaller than Healey's, but such a duct was observed in nearly 50 per cent of cases.

The legend of the hepato-cholecystic bile-duct presumably owes its origin to the subvesical duct. In fact, no case of a duct emerging from the gall-bladder bed of the liver and actually entering the gall-bladder was found either by Healey, or by the present writer. Were it proved to occur, the connexion would then be of great theoretical importance as the only known example of anastomosis between separate branches from the common bile-duct.

The gall-bladder bed is not the only area where bile-ducts have been described to leave the liver. The so-called 'aberrant ducts' were first described by Ferrein (1733), and later by Kiernan (1833). Extrahepatic bile-ducts (other than the main trunks) are frequently to be found in the connective tissue that surrounds the inferior vena cava where it is buried in the back of the liver, and in the extraperitoneal fat between the layers of the falciform ligament near its liver attachment, and also in the tissue bridging the fissure for the ligamentum teres and the saccus. Staining in these areas has often been noted after injection along the common hepatic duct of a dye solution.

Surgically, however, the most interesting group are those which leave the left extremity of the liver to course for a varying distance between the layers of the left triangular ligament, perhaps even reaching the diaphragm. An example of this condition is shown in *Fig.* 759 where the extrahepatic bile-ducts, filled with gelatin by injection of the common hepatic duct, are clearly seen. They are accompanied by portal vein branches, which have been filled with a gelatin-carbon suspension, and microscopy showed that prolongations of intrahepatic arterial branches were also present. The frequency of the condition is assessed most variably by different authors, but it cannot be very uncommon as it was found here in no less than three of the twenty specimens which included an umbrella of diaphragm, and in which the search was specifically made. Surgical piquancy has been lent to the original anatomical descriptions by a recent publication (Rapant and Hromada, 1950) describing 2 cases where mobilization of the left lobe of the liver was followed by leakage of bile from abnormal ducts in the triangular ligament.

Resections of Liver.—The recognition that liver architecture is segmental, and that the intrahepatic channels follow reasonably constant courses, may bear on the surgery of the organ in three possible ways. First, it makes possible the planning of incisions and excisions in such a manner that no portion of liver tissue meant to be left in situ is deprived of blood circulation or biliary drainage. Second, it raises the possibility that there may exist planes which are relatively bloodless, and so peculiarly suitable for use by the surgeon. It must be realized that these two are entirely separate considerations. Third, it implies the feasibility of segmental resection assisted, as regards the control of hæmorrhage, by extrahepatic ligation of the vascular pedicles.

Planned Excisions.—This phrase is of wide scope. It implies that whenever the surface of a liver is broached, the surgeon should visualize the arrangement of the hollow structures in that region, and should be able to assess how much derangement of liver structure, and therefore function, he may be causing. In particular, it is important that he should remember the possibility of causing disturbance at some distance from the site at which he inflicts his injury.

These considerations apply to local excisions, and also to the trimming of lacerations of the organ. The most obvious examples are that an incision into the upper part of the right or the left lobe is likely to interrupt the appropriate hepatic vein and so destroy the venous drainage from the lower part of the lobe. In the present state of our knowledge of hepatic vein anastomoses, it would therefore appear unwise not to excise the lower part of the lobe as well, by extending the incision completely across the lobe.

As regards '*bloodless planes*', the main proposals have been the principal plane, the plane of the falciform ligament, and the plane subdividing the right lobe. Reference to a cast in which all the hollow structures have been injected (*Fig.* 751) warns how small is the gap in any area between sizeable branches of neighbouring Glissonian segments. It follows that the essential prerequisite of any bloodless plane is that it be accurately identifiable from the surface and at all times as the incision proceeds.

On this count, it has already been shown that the plane subdividing the right half is most unsuitable. It is variable in location, irregular in contour, not sharp near the anterior liver surface, and it does not necessarily contain the right hepatic vein.

The plane of the falciform ligament, however, is almost ideal. It is very clearly demarcated on the surface, anteriorly by the attachment of the falciform

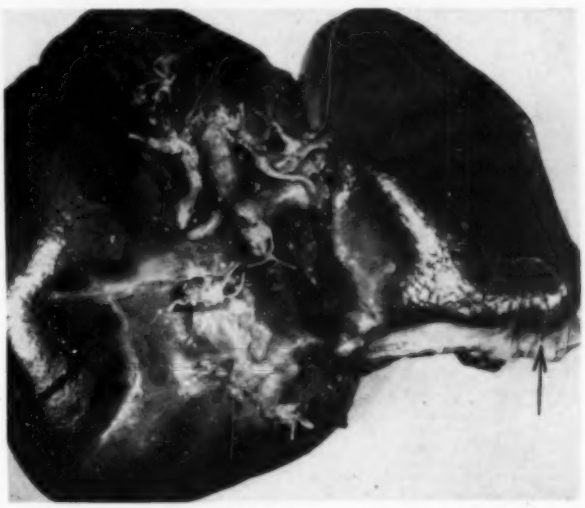

FIG. 759.—The liver has been turned upwards. Bile-ducts leaving the liver at its left pole to course along the left triangular ligament are arrowed.

ligament and posteriorly by the fissure for the ligamentum teres and the groove between caudate lobe and left lobe. Only a few vessels cross the plane in its lower third. Moreover, the liver at this site is usually quite thin and so bleeding into the incision is relatively easily controlled by digital pressure, a method recently emphasized by Brunschwig (1955). Later, through-and-through interlocking sutures are inserted.

The third possibility, the principal plane, is less satisfactory. Posteriorly, the groove along the middle of the gall-bladder bed is an accurate guide to the plane, but elsewhere there is no visible surface marking, and the caudate lobe must be bisected by eye. Anteriorly, a line drawn from the left side of the inferior vena cava at the upper margin of the liver to meet the apex of the gall-bladder notch marks the plane fairly well, but local departures from the line are common and one is shown in *Fig.* 750. Again, the thickness of the liver is approaching its maximum in this region, and hæmostasis is correspondingly more difficult than in the plane of the falciform ligament. However, it would appear that splitting the liver along this plane would be at least greatly facilitated by the anatomy, and, as Couinaud (1955) suggested, the procedure may be useful in the approach to the porta where for one reason or another the conventional route from the back is contraindicated.

Assisted Segmental Resection.—The first description of a partial hepatectomy with precognition and control of the blood-supply and biliary drainage to the part resected was given by Pettinari (1947) and independently by Raven (1949). These authors performed ablation of the left lobe; that is, up to the plane

of the falciform ligament. Lortat-Jacob and Robert described right lobectomy in 1952, and publications describing the same and similar operations have followed at an ever-increasing frequency. A new era of hepatic surgery has dawned.

When the blood-supply is ligated, colour changes are said to occur in the affected segment. Guidance is thereby given as to the correct line of section, and hæmorrhage is reduced.

The possible operations follow directly from the anatomy described previously. There are two main

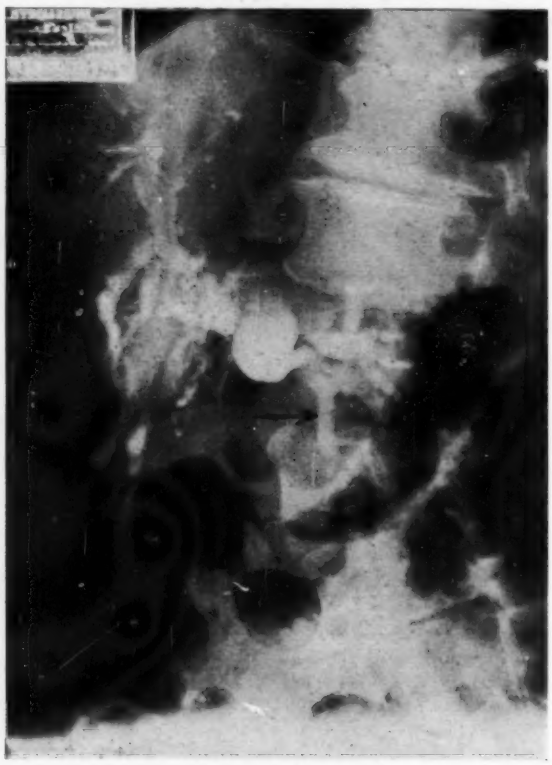

FIG. 760.—Transhepatic cholangiogram of a case of obstructive jaundice, showing a large duct draining the quadrate lobe (arrowed).

groups: *formal hepatectomy*, and *semi-formal*. In the former case, both the Glissonian and the hepatic vein pedicle to a segment are controlled ; in the latter, only some elements of the vascular pedicles can be tied.

In the present state of our knowledge, there are only two operations which can properly be designated *formal*: they are *right and left lobectomy*, the removal respectively of the classical right and left lobes, the plane of section being in either case that of the falciform ligament. The left lobe is supplied by the left hepatic vein, and all branches of the Glissonian system originating at the left border of the saccus; the right by middle and right hepatic veins, and by the right division of the portal vein and all branches of the left division given off before the saccus, or from the right border of the saccus (*Fig.* 753).

The *semi-formal* hepatectomies vary in the extent of aid that they receive from the anatomy, and correspondingly in their ease of technical execution. Excisions based on the principal plane may properly be called *right and left hemi-hepatectomy*. The corresponding hepatic vein may be secured, but until more is known about the degree of functional anastomosis between the hepatic veins, it would seem unwise to tie the central hepatic vein in either operation. It will be remembered that this central vein lies in the principal plane itself. In performing a left hemihepatectomy, removal of the central vein might endanger the anteromedial zone of the right half of the liver and so the vein should be preserved and the section made slightly to its right. Similar considerations apply to right hemihepatectomy.

In performing the hemihepatectomies, a very important point to remember is that the biliary branch to the posterolateral zone of the right half, or less often that to the anteromedial zone, may originate from the *left* hepatic duct (*Fig.* 755). In such a case it would be easy for the surgeon mistakenly to think that the junction of the first right branch with the common hepatic duct was really the bifurcation of the common duct. Performing a left hepatectomy, he might then tie and divide the main duct immediately above the first right branch, and so destroy the biliary drainage of half the liver remnant. It is essential, therefore, to follow the Glissonian structures well to the left (or right, as the case may be) before dividing them.

Excisions of smaller masses of liver than the above cannot be aided by tying a hepatic vein. In the *left lobe*, Patel and Couinaud (1955) suggest the possibility of excision of the inferior half alone, and certainly the appropriate Glissonian pedicle can be ligated. However, the left lobe itself is usually of quite small volume, so that it is difficult to imagine circumstances when left inferior segmentectomy, with its minor saving of hepatic tissue, should be preferred to left lobectomy, with its clearly demarcated plane in which hæmorrhage is easily controlled. In the *right half* the subdivision, these authors propose, of the two zones by a horizontal plane into four segments can be ignored, because the anterosuperior and posterolateral bile-ducts, arteries, and portal veins do not subdivide until they have plunged a considerable distance into the liver, and so are not accessible at the porta without incision into liver substance. Excision of one or other zone alone is theoretically possible, but the properties of the plane between the zones have been shown to be inimical to surgery. It is important to remember, too, that though the bile-duct to the posterolateral zone usually springs from the common duct at a higher level than that to the anteromedial zone, the opposite arrangement may also occur. There would then be a chance of ligation of the bile-duct to the wrong zone. Despite all these difficulties, however, such operations have been performed, for example by Fredet and Bernier (1956), but they owe much less to anatomical niceties than to the technical dexterity of the surgeons concerned.

Another operation based partly on the right innominate plane is that described by Gans (1955 a) as 'middle lobectomy'. The left limit of resection is the plane of the falciform ligament, and a truncated wedge of tissue bounded by these two planes is removed right back to the porta hepatis. The

now-isolated left lobe and right posterolateral zone are united by capsular stitches. Before the ablation the central hepatic vein and the appropriate Glissonian pedicles are tied. This operation also suffers from the deficiencies of the right interzonal plane. Gans advocated it for cancer of the gall-bladder, but on the basis of his own description of the mode of spread of the growth into the liver, it would appear that the more radical, yet technically easier, operation of right lobectomy would offer a much greater chance of cure.

Removal of caudate or quadrate lobes, separately or together, has not been investigated in detail.

Intrahepatic Cholangiojejunostomy.—One result of investigating internal structure is that it raises the possibility that a particular intrahepatic bile-duct can be found with ease and regularity, and used for draining into intestine an irremediably-blocked biliary system. The utility of such procedures lies mainly in cases where the obstruction is non-malignant, and often traumatic and the result of previous surgery. Longmire states (Lipmann and Longmire, 1954) that benign stricture of the common hepatic duct rarely reaches so high as the junction of right and left hepatic ducts, and therefore the whole liver can usually be drained of bile through a duct in one half or the other.

It was Longmire who first published a method, and some examples of its use (Longmire and Sanford, 1948). An incision was made into the left lobe of the liver from the anterior surface, and carried straight backwards until there appeared on the cut surface the end of a transected bile-duct which, because of the effects of back-pressure, had dilated to a calibre that made it suitable for anastomosis. The section of the left lobe was then continued to the posterior surface with amputation of a broad wedge of the left lobe, and the end of the duct implanted in a loop of jejunum.

No precise instructions are given in the paper as to the exact location of the plane of section, but the photographs indicate a point two or three finger-breadths to the left of the plane of the falciform ligament. The section therefore follows no anatomical plane and would be likely to provoke serious hæmorrhage. The large duct found would be either the superior or the inferior duct to the left lobe.

Dogliotti, however, had previously, in 1946, performed a similar operation which appears to take greater advantage of the anatomy, but did not describe his operation till after Longmire's work was published (for example, *see* Dogliotti and Fogliati, 1954). He used the stomach for his anastomosis, a procedure which can be criticized: far more important, however, he cut right across the liver *in the plane of the falciform ligament*, and isolated what he terms the 'central bile-duct of the left lobe' at the cut surface. Often one or other of the superior and inferior left lobar ducts is larger than its fellow, or else the main left duct subdivides late so that it is still present as a single large duct at the plane of the falciform ligament, and this is the canal Dogliotti presumably means.

There is yet another possibility: one that would obviate the necessity for sacrificing a large portion of liver. The consideration is important, because in the nature of the problem the liver is already taxed by the back-pressure in its obstructed bile-passages. The quadrate lobe, as has been seen, is drained by one or a few bile-ducts, rarely more than two. The sole duct, or the left-most of multiple ducts, runs downwards and forwards from the porta hepatis towards the inferior margin of the liver, and usually in the depths of the fissure for the ligamentum teres or in the right wall of the fissure. It is

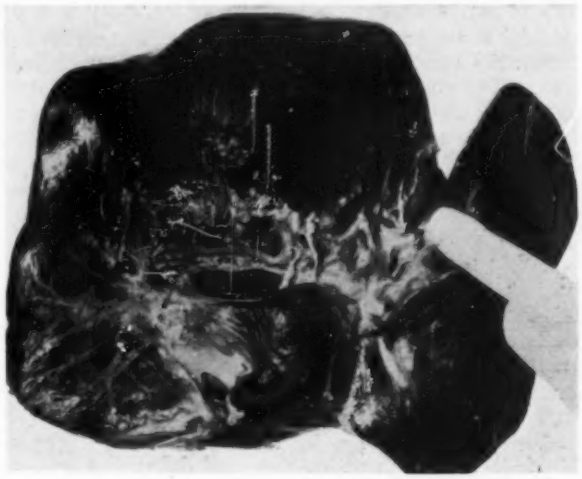

FIG. 761.—The liver turned upwards. The finger holds aside the left edge of the fissure for the ligamentum venosum, which has been deepened by blunt dissection, and points at a bile-duct to the quadrate lobe running forwards in the fissure.

suggested that the operation be commenced with Dogliotti's procedure in mind, but that section should commence at the inferior border of the liver and proceed (by blunt dissection) on the posterior surface in the fissure. In this way, the duct to the quadrate lobe may be encountered. It is always quite easy to find in a post-mortem liver, and though it is sometimes of a rather narrow diameter, perhaps as little as $\frac{1}{10}$ in., it is much more frequently of important proportions. Often large enough even in the absence of biliary obstruction to be anastomosed to intestine, there is every likelihood that it would prove suitable in the type of case here being considered (*Figs.* 753, 761). There is some evidence to support this view. *Fig.* 760 shows a transhepatic cholangiogram in a case of jaundice due to carcinoma of the head of the pancreas: the duct to the quadrate lobe is clearly outlined, and very nearly as large as the main ducts to the left lobe. (Nurick, Patey, and Whiteside, 1953, described this case, though this particular radiograph was not published in their article.)

Supposing the operation to prove impossible, because of a particularly small duct to the quadrate lobe, the section along the plane of the falciform ligament can be completed (with the aid of extra-hepatic ligation of the blood-vessels if necessary), and a large duct to the left lobe used for the anastomosis, as Dogliotti described.

One important relation of the duct to the quadrate lobe must be remembered. If one dissects upward along the fissure for the ligamentum teres, one will ultimately meet the blind termination of the left portal vein, which has turned forwards along the fissure. The duct lies deep (i.e., anterior) to this saccus, and only

where the saccus ends does it gain superficiality (*Figs.* 753, 761). The saccus rarely being much more than 1 in. long, it follows that efforts to locate the duct will be safe, provided they are pursued to no nearer than 2 in. from the porta hepatis and the transverse part of the left portal vein.

SUMMARY

Various methods of investigation of intrahepatic structure are described, including the injection of coloured dyes along separate bile-ducts.

The usual patterns of the four systems of hollow structures are discussed, and the validity of subdivision of the organ by means of certain avascular planes, as described by previous authors, is tested by the dye method, and found wanting except in two cases. These planes are here called the *principal* plane, and the *plane of the falciform ligament*. Respectively they divide the liver into *right and left halves*, and *right and left lobes*.

After reference to superficial and accessory bile-ducts, which may be in danger during operations on neighbouring structures, the principles underlying resections of liver substance are discussed, and the conclusion drawn that only right and left lobectomy and right and left hemihepatectomy, derive directly from anatomical considerations.

Finally, a new operation, involving isolation of the duct to the quadrate lobe, is described for the relief of high obstruction of the common bile-duct.

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SIMPLIFIED HEPATECTOMY

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DURING the last two decades notable progress has been achieved in anæsthesia, surgical physiology, and chemotherapy, which has enabled surgeons to enlarge the scope of surgical treatment with lower operative mortality and morbidity. For instance, hepatic resection, once the monopoly of surgical giants, has been an operative procedure that many surgeons having the necessary environmental amenities can undertake without much hesitation.

It is one of the fundamental concepts of surgical therapy that when dealing with malignant growths of viscera it is important to resect adjacent structures involved by the malignant process. This is especially true in cancer of the cardia of the stomach, in which condition it is often difficult to make a positive diagnosis despite meticulous examination by radiography, endoscopy, etc. Not infrequently surgeons encounter more or less advanced carcinoma originating in the anterior aspect of the cardia which often invades the left hepatic lobe by direct infiltration. Under such circumstances, and where there are no metastases in non-resectable structures, resection of the liver should be formally carried out as a part of the operation, as advocated by Wangensteen (1951) and others, thus increasing the scope of operability.

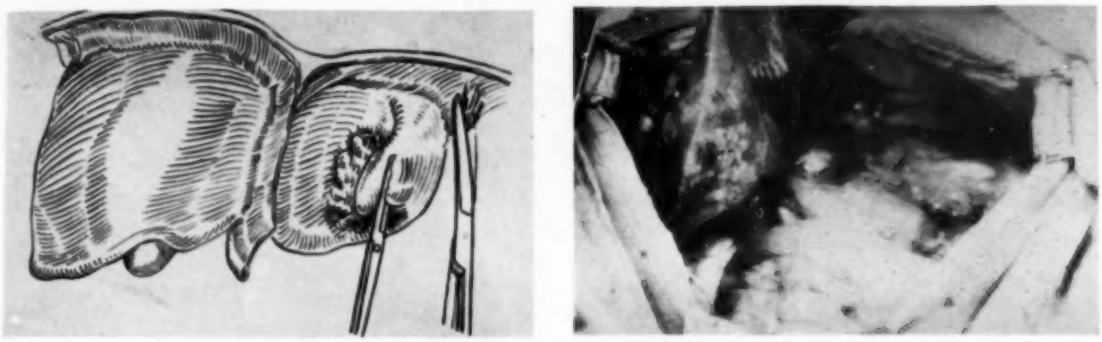

FIG. 762.—Mobilizing the left lobe of the liver.

A number of techniques for liver resection have already been published (*Table I*). Nevertheless, all those which are in use at the present time demand complicated manœuvres which from the practical standpoint seem far from being finalized. Moreover, most procedures are limited to solitary resection of the liver, and some of them are too theoretical to be resorted to in practice. In general, there is in most reports a common trend to employ large numbers of sutures and a variety of packs as a means of circumventing uncontrollable hæmorrhage, and this seems to be the major deterrent to most surgeons from operating on the liver, yet there is ample evidence that bleeding is far from being insurmountable. In such operations, the risk of infection or biliary leak may be overcome by special attention to drainage, admittedly complicating the operative procedure.

Table I.—PRINCIPLES OF DIFFERENT TECHNIQUES

(*After P. W. Stone*, 1951)

1. Compression of the pedicle of the tumour by elastic ligature and extra-peritonealization (Cousins, 1874).
2. Cautery excision of tumour (Escher, 1886).
3. Lobectomy of the lobe containing the tumour (Langenbuch, 1888).
4. Introduction of preliminary hæmostatic sutures, tying them after resection (Kousnetzoff and Pensky, 1896).
5. Placing of clamps on cut edge, exteriorization of clamps and removal at a later date (Clementi, 1890).
6. Gauze tamponade of cut surface (Anschutz, 1903).
7. Excision and hæmostasis by electrocautery (McLean, 1929).
8. Excision and hæmostasis by electrocautery following preliminary placement of hæmostatic sutures (Reid, 1945).

FIG. 763.—The blades are ovoid in cross-section, and the beak-like end of the under-blade prevents liver from being squeezed out beyond the tips.

The purpose of this report is to present a simplified method of liver resection developed by the author, and especially as part of the ablation of a malignant process originating in an adjacent organ without fear

of either hæmorrhage or biliary drainage during the post-operative course. The author will herein touch only a few details of this operative method.

OPERATIVE METHOD

The method is preferably employed for resection of the left lobe, whether partial or total, the caudate or quadrate lobe, or a part of the right lobe, the necessity for which surgeons not infrequently encounter. The technique, however, is not suitable for massive resection of the right lobe.

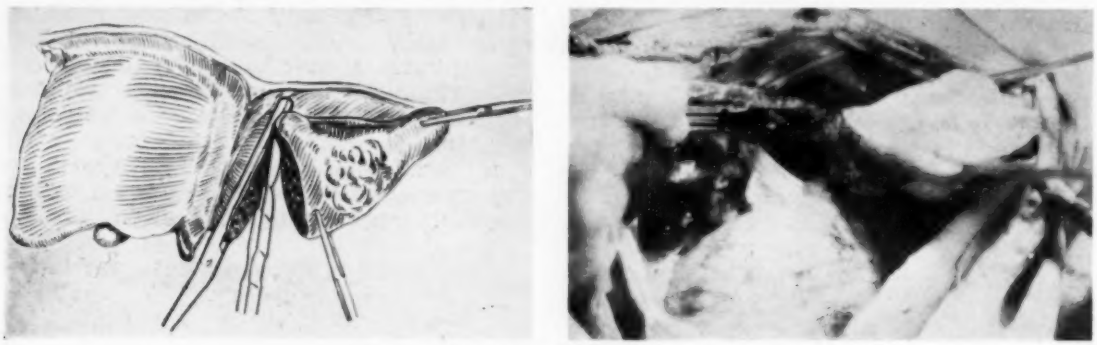

FIG. 764.—Excision of the left lobe of liver.

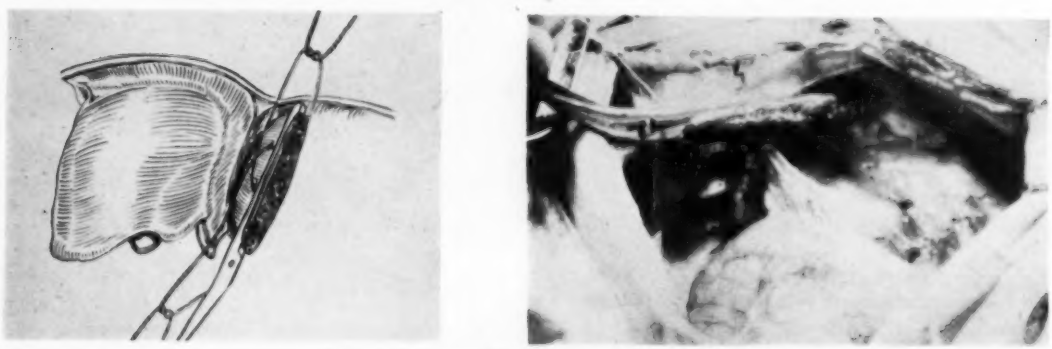

FIG. 765.—Suturing raw surface of liver.

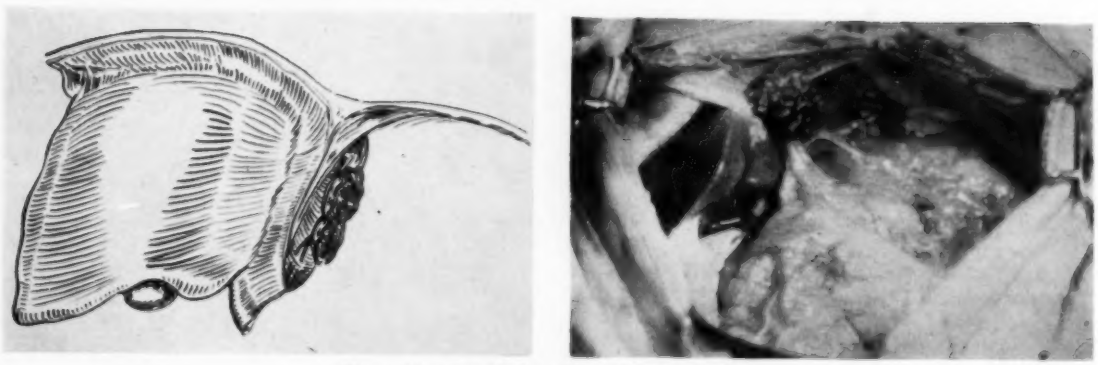

FIG. 766.—Completion of insertion of sutures.

1. Resection of the Left Hepatic Lobe.—The operation is commenced by a high midline incision. The ligamentous attachment between the diaphragm and the left lobe is incised towards the diaphragmatic hiatus (*Fig.* 762). Beyond the hiatus the severance is extended to the right crus of the diaphragm, after which procedure the left lobe can readily be rotated forwards and slightly to the right. Then, the author's crushing clamp, as shown in *Fig.* 763, is placed across the intact portion of the left lobe. After crushing, the lobe is severed by a sharp knife (*Fig.* 764). The gradual closure of the clamp is sufficient to gain a complete crushing effect on the parenchyma along the blade of the clamp, the instrument being so constructed that the parenchyma alone is crushed, the capsule being left intact. Resection is performed about 0·5 cm. beyond the clamp. The next step is the treatment of the remainder of the lobe. A few ligatures are employed for the large blood-vessels appearing on the bare surface, and two or three large sutures are passed through the entire thickness of the lobe along the inside of the blade of the clamp (*Figs.* 765, 766). The ends of the sutures are then tied very gently as the clamp is gradually removed. Needless to say, where the resection is small, fewer sutures will be necessary, sometimes only one.

Neither packs of gauze or Gelfoam, nor autografts of greater omentum or peritoneum are needed for treatment of the bare surface, and the use of the cautery is rarely required. In actual fact experimental and clinical experience at our clinic shows that there is little risk of parenchymatous hæmorrhage or biliary drainage following this operative procedure.

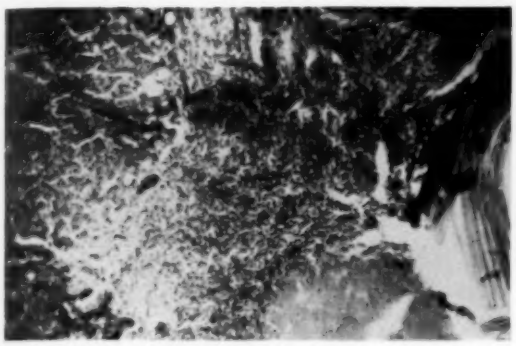

FIG. 767.—Necrotic area in suture line.

After completing the operation, a polyethylene film drain is placed in the same side of the upper abdominal cavity, and is usually withdrawn by ten days after operation, though it depends on the amount of drainage.

2. Resection of the Right Hepatic Lobe.—Hitherto, removal of a solitary tumour located in the right lobe was carried out by the wedge form of resection as practised by Zweifel-Payr, Kessenetzoff, and Pansky. Use of the author's clamp makes the procedure much shorter, since the troublesome conventional manœuvre of suture is no longer necessary.

HISTOLOGICAL STUDY

An important feature in the assessment of the value of this simplified hepatectomy is to observe the healing process in the stump of the remaining gland. Histological study of cases coming to autopsy provided valuable information regarding this matter. Twenty-five days after operation there was marked infiltration by fibroblasts and round cells growing centripetally into the necrotic areas produced by the ligatures near the stump, and fatty infiltration was scattered throughout the neighbouring portion (*Fig.* 767). Four months later this reaction was replaced by a thick layer of connective tissue associated with fibroblastic thickening of Glisson's capsule

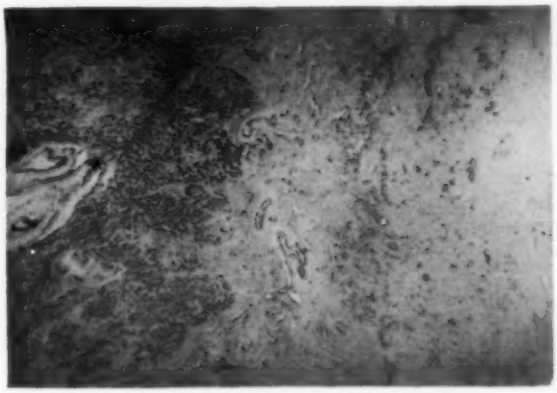

FIG. 768.—Histology at end of four months.

as well as cicatrical atrophy of the neighbouring liver tissues (*Fig.* 768). In the light of these facts, one may assume that the repair of the bare surface is complete within four months of the operation.

OPERATIVE RESULTS

The total number of hepatic resections undertaken at our clinic by the end of April, 1956, was 45, of which 6 cases underwent liver resection alone, whereas in the remaining 39 cases the hepatectomy was part of a combined resection of a neighbouring viscus, as shown in *Tables II* and *III* respectively. No deaths occurred in the group of solitary hepatectomy. The cause of death in 2 cases after combined resection, involving more than four organs, was emphysema thoracis due to a leak from the anastomotic site in the gut. No deaths were attributable to the hepatectomy itself. The same technique was employed for gastric ulcers penetrating into the liver, where resection *en bloc* of liver and ulcer-bearing part of the stomach was easier than separation from the ulcer; some of these ulcers were malignant.

Table II.—DETAILS IN CASES WITH LIVER RESECTION ALONE
(Nakayama Surgical Department, up to end of April, 1956)

NAME (Initials)	SEX	AGE	OPERATIVE DIAGNOSIS	REMOVED PORTION	PATHOLOGICAL DIAGNOSIS	END-RESULTS
H. Y.	Male	43	Hepatoma	Subtotal lt. lobe (135 g.)	Tumour due to ascariasis	8 yr. 2 mth. (alive)
K. O.	Male	63	Hepatoma	Total lt. lobe (360 g.)	Hepatoma	3 yr. 6 mth. (alive)
S. M.	Male	52	Liver gumma	Total lt. lobe (459 g.)	Liver Tb.	2 yr. 10 mth. (alive)
Y. T.	Female	52	Liver cyst	Total lt. lobe (350 g.)	Liver distoma	2 yr. 2 mth. (alive)
T. I.	Male	68	Hepatoma	Total lt. and caudate lobes (570 g. and 300 g.)	Cholangiogenous carcinoma	4 mth. (died)
N. A.	Female	13	Liver tumour	Subtotal rt. lobe (350 g.)	Hepato-adenoma	1 yr. 7 mth. (alive)

FOLLOW-UP RESULTS

1. Hepatectomy Alone (*Table II*).—Of the hepatectomized patients, all except one case of carcinoma of the bile ducts are still alive and well, the longest survival period being over eight years. This group includes one four-year survivor with a hepatoma who follows his usual occupation free from any sign of recurrence.

2. Combined Resection (*Table III*). — As opposed to benign lesions this group of malignant cases gave rather poor results. It must be borne in mind, however, that most of those malignant cases included in this group were so advanced that recurrence was inevitable, yet we have one 3-year survivor. It is our view that, since surgical removal is still the only real cure for malignancy at the present time, efforts must be made to resect *en bloc* the liver involved, if practicable.

FIG. 769.—Cut surface of hepatoma.

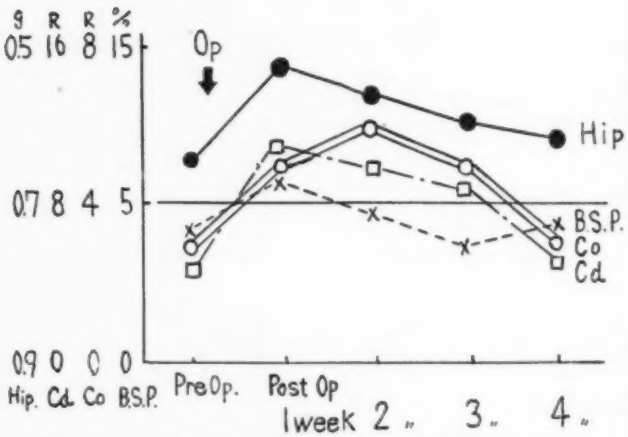

FIG. 771.—Recovery of liver function after solitary liver resection.

Table III.—DETAILS IN CASES WITH COMBINED RESECTION OF THE LIVER

Organs Resected en bloc	*No. of Cases ( ) indicates Ulcer*	*Operation Deaths and Mortality Rate*
Entire stomach, liver, spleen, tail of pancreas, colon, and left suprarenal gland	2	1 (50 per cent) (by leak at Anastomosis)
Entire stomach, liver, spleen, tail of pancreas and left kidney	1	0
Entire stomach, liver, spleen, and tail of pancreas	4	1 (25 per cent) (by leak at Anastomosis)
Entire stomach, liver, and spleen	2	0
Entire stomach and liver	13 (1)	0
Cardia, liver, spleen, and tail of pancreas	1	0
Cardia and liver	6 (1)	0
Stomach and liver	5 (2)	0
Gall-bladder, liver, and colon	1	0
Gall-bladder and liver	2	0
Colon and liver	2	0
TOTAL	39 (4)	2 (5·13 per cent)

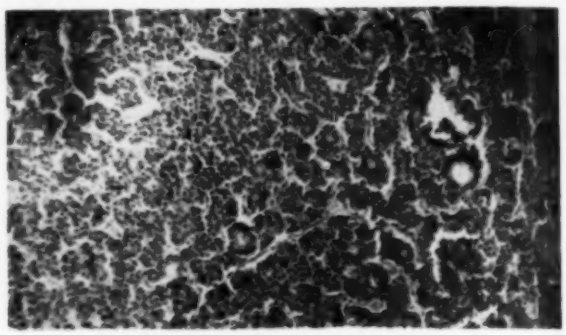

FIG. 770.—Histology of hepatoma.

CONCLUSION

Although various methods of hepatectomy have their different advocates, the advantages of the one described by the author are: (1) Feasibility, since the use of as simple an instrument as a crushing clamp is all that is needed to obviate complicated manœuvres; and (2) There is no fear of untoward complications such as hæmorrhage or biliary drainage, as our considerable clinical experience amply proves. We believe that this simplified method of liver resection is worthy of trial, especially where a combined resection is called for, yet, at the same time where it is desirable to avoid prolonging the operation time.

CASE REPORTS

Case 1.—Japanese male, aged 63 years, was seen at the Nakayama Clinic because of a mass in the epigastrium and symptoms of fullness of the stomach and occasional pain. A large tumour, smooth on the surface, could be seen in the epigastrium, moving with respiration. Physical examination was otherwise normal. Examination of the blood: R.B.C.s 4,250,000 per c.mm.; W.B.C.s 6800; Hæmoglobin 98 per cent (Sahli). No abnormalities in the urine. Occult blood negative. Radiography of the chest normal; of the gastro-intestinal tract showed an immobile area along the lesser curvature of the stomach. The most probable diagnosis was carcinoma of the pylorus, and exploration was carried out on Oct. 8, 1952, under spinal anæsthesia. The abdomen was opened through a high mid-abdominal incision. There was no ascites and the stomach and spleen were normal, but there was an egg-sized, pale-yellowish, firm tumour in the middle part of the left hepatic lobe. The specimen when removed weighed 360 g., and measured 16 × 8·5 × 6·2 cm. When transected it was firm and white (*Fig.* 769). Pathological diagnosis: Hepatoma (*Fig.* 770). After an uneventful post-operative course he was discharged 22 days later. A speedy recovery in liver function was noted (*Fig.* 771).

Case 2.—Japanese male, priest, aged 55 years, was admitted to the Nakayama Clinic for surgical exploration. Symptoms were epigastric discomfort for six months, changing to periodic pain for one month. An egg-sized, tender, elastic tumour was noticed, moving with respiration, slightly on the left of the epigastrium. Abdomen otherwise was normal. R.B.C.s 2,380,000; W.B.C.s

FIG. 772.—Operation specimen.

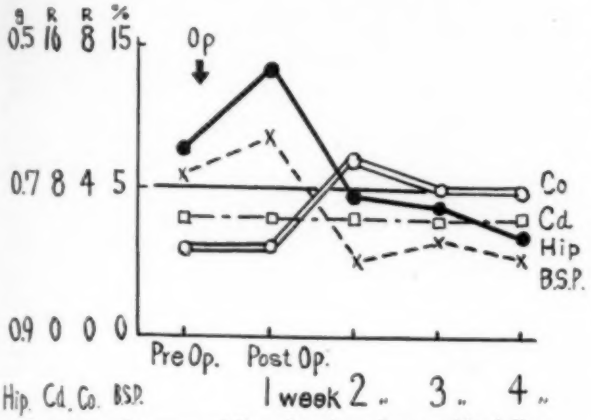

FIG. 774.—Recovery of liver function after combined liver resection.

6800; Hæmoglobin 55 per cent. Occult blood was strongly positive. Barium meal revealed cancer of the stomach.

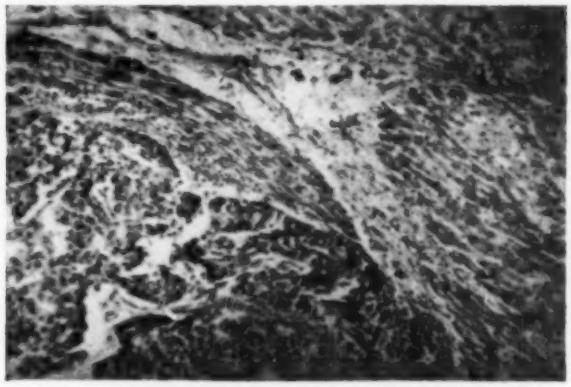

FIG. 773.—Histology of removed tumour (Case 11).

Laparotomy was performed on Dec. 15, 1954, under spinal anæsthesia. A tumour the size of a fist was found on the lesser curvature, with many metastases in the splenic and other nodes, and direct infiltration of the liver was observed. Total gastrectomy combined with resection of the left hepatic lobe, the spleen, and the tail of the pancreas was carried out (*Fig.* 772). Microscopical structure of the lesion is shown in *Fig.* 773. The liver function returned to normal within four weeks (*Fig.* 774).

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RESECTION AND RADICAL AMPUTATION IN THE TREATMENT OF TUMOURS OF THE BONY PELVIS, ILLUSTRATED BY A CASE OF PUBIC RESECTION

BY ROBERT MONRO, IPSWICH

OF recent years hindquarter amputation has become established as the treatment of choice in the majority of cases of osteogenic tumour of the pelvis (Gordon-Taylor and Monro, 1952; Gordon-Taylor, Wiles, Patey, Turner-Warwick, and Monro, 1952). None would deny, however, that it is a major mutilation, and in certain circumstances resection of only a segment of the pelvic girdle may be the elective procedure and need not result in any disability.

CASE HISTORY

In 1952 G. J., aged 21, an electrician, at that time serving with the Fleet Air Arm, gave a history of five months of occasional pain in the lower abdomen and right groin. There was no discernible external abnormality, but on rectal examination a hard nodular tumour 4 cm. in diameter was palpable, arising from the back of the pubis and abutting on the prostate and base of the bladder. Radiographs showed a partially calcified tumour projecting from the back of the pubis and indenting the base of the bladder (*Fig.* 755). This was thought to be cartilaginous, and in view of some seeming rarefaction in the adjacent pubis, it appeared likely to be sarcomatous (Monro, 1953).

The advisability of hindquarter amputation was considered, but it was decided that, with the tumour situated as it was, a local resection offered as much chance of effective clearance as did a hindquarter amputation, and yet entailed much less mutilation.

AT OPERATION.—On Sept. 17, 1952, an incision was made corresponding to the anterior part of the hindquarter amputation, extending from mid-iliac crest above the inguinal ligament to just beyond the midline above the pubis and allowing access for temporary control of the common iliac artery (Gordon-Taylor and Monro, 1952). A second incision was carried back from the pubic tubercle to the ischial tuberosity in the perineal crease. The greater part of the right pubis was then resected with the tumour attached posteriorly, the latter at all points appearing to have a covering of normal tissues. The wound was closed in its lateral part with interrupted thread stitches, but a considerable gap was left in the medial part of the wound in the position formerly occupied by the body of the pubis.

Fears were entertained that an awkward hernia might develop, and though Muir (1951) had reported shortly beforehand that this gap had occasioned no disability in a woman on whom he had carried out a similar resection, the gap was closed as a precautionary measure with a double-layered sheet of tantalum gauze (*Fig.* 776).

FIG. 775.—Radiograph of pelvis, showing the partially calcified tumour projecting internally from the back of the pubis with some apparent rarefaction of the adjacent pubis.

FIG. 776.—Radiograph of pelvis, showing the extent of the resection, and the gap in the girdle filled in with tantalum gauze.

FIG. 777.—Patient balancing on right leg after resection of right pubic bone.

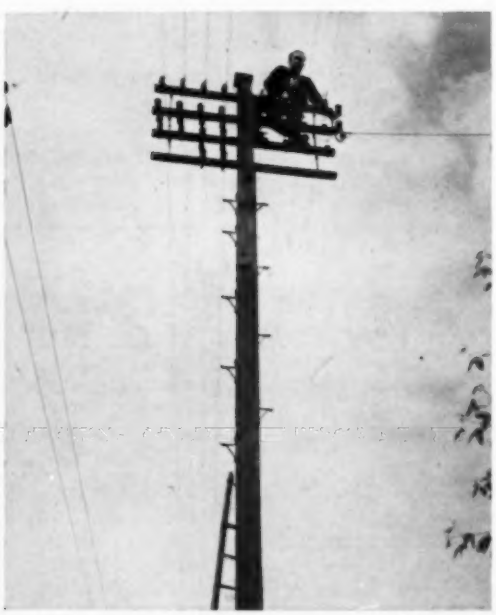

FIG. 778.—Patient at work on the top of a telegraph pole.

THE SPECIMEN.—This comprised the body of the pubis with superior and inferior rami, and arising from its posterior surface, a nodule of cancellous bone 4 × 3 × 2·5 cm., capped with a thin covering of cartilage. Histological examination confirmed that this was an exostosis with no hint of malignancy.

PROGRESS.—No attempt was made to immobilize the limb post-operatively. Full movement at the hip was noted on the tenth day. The patient was sitting out of bed on the fourteenth day and started walking on the eighteenth, albeit with some feeling of stiffness. After four weeks he was walking well and negotiating stairs with ease. The wound healed soundly and has never occasioned the least disability. Four years after his operation there is slight bulging in the region of the right pubic body and a slight cough impulse, a state of affairs comparable to the condition known as Malgaigne's bulge, but there is no definite rupture. There has been no recurrence of the tumour.

The patient was invalided from the Navy and took employment as an electrician in the Post Office, being concerned with outside maintenance, calling for a high degree of strength in handling telegraph poles (*Fig.* 777) and agility in climbing them (*Fig.* 778). Subsequently the patient rejoined the Services, this time in the Army. He completed his battle training successfully and has become a tank gunner. He was last seen in September, 1956, four years after his operation, on embarkation leave and about to sail for active service in the Middle East.

DISCUSSION

It has been the experience of a number of surgeons that resection of the pubis causes little disability (Muir, 1951; Young, 1956), either in respect of locomotion or herniation, and yet such resection may hold prospect of eradicating a tumour successfully. Even more massive resections involving the acetabulum and calling for extensive orthopædic reconstruction, but giving correspondingly poorer function, have been carried out (Lipscomb, 1952), though it would seem that if the extent of the tumour within the os coxæ is such that its resection entails disorganization of the hip, it is probably inviting recurrence not to do a hindquarter amputation.

The case here described is an outstanding demonstration that the pubis can be resected to eradicate a tumour without occasioning any disability whatever. It also illustrates certain problems in relation to diagnosis in pelvic tumours and exemplifies points to be assessed in deciding between hindquarter amputation and conservative resection.

It might be thought that a biopsy would have been of value in this instance, for pre-operatively the tumour was considered to be a chondroma, probably a chondrosarcoma, whereas in fact it proved to be an exostosis. Nevertheless, it was to be remembered that cartilaginous tumours have a well-established tendency to local recurrence unless removed with an intact investment of normal tissues, and biopsy was to be deprecated. This outlook on biopsy is particularly important with tumours projecting into the pelvis or abdomen, where there is no chance of resection at a more proximal level if recurrence does take place. In such tumours, therefore, the least procedure which should be contemplated should be a block resection, and if any form of preliminary exploration is undertaken, this should be merely to assess the extent and operability of the tumour and should be made transperitoneally, through a remote incision, preserving the integrity of tissues over the internal aspect of the tumour until definite resection or amputation. It should also be pointed out that biopsy, apart from being dangerous, may be misleading, and in this instance would probably have been so, for the exostosis had a cartilaginous cap whose microscopical appearance without the main mass of the exostosis would have confirmed the clinical impression that the tumour was a chondroma and done nothing to dispel the suspicion that it was a chondrosarcoma, since cartilaginous tumours may not be homogeneous and may contain well-formed cartilage in one area and obvious malignant tissue in another (Jaffe, 1947).

It has previously been suggested that the outlook on treatment of osteochondral tumours of the pelvis should differ if the tumour is on the outside of the pelvis or within (Monro, 1953). When a tumour arises externally on the pelvic girdle, there should be extreme reluctance to do anything other than a hindquarter amputation, for resection may invite recurrence and in breaching the integrity of the pelvis invites recurrence in situations no longer accessible to such amputation.

The case recently described by Young (1956), where amputation was advised but declined and a local resection perforce carried out, amply illustrates this point. Recurrences later developed in the abdominal wall and retroperitoneally.

Where, however, a tumour arises internally and is small and accessible to local resection, and yet, by reason of the proximity of the tumour to viscera and peritoneum, hindquarter amputation offers little more in terms of effective clearance than block resection, then resection without biopsy should be performed. It may offer as good a prospect of survival and ensures incomparable superiority of form and function.

SUMMARY

A case of resection of the pubis for osteochondroma is described and attention drawn to the excellent function which may be retained following this operation.

The points to be considered in deciding whether to carry out a resection of the pelvic girdle or a hindquarter amputation in tumours of this neighbourhood are discussed.

Comment is made on the apparent paradox that there should be an inclination to treat tumours on the internal aspect of the pelvis by resection, and those on the external aspect by hindquarter amputation.

Acknowledgement.—Though this patient has been followed up at Ipswich, his operation took place on the Surgical Unit at The Middlesex Hospital, and my thanks are due to Mr. D. H. Patey, Director of Surgical Studies, who has kindly agreed to my publishing these observations.

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SIMPLE ULCER OF THE SMALL INTESTINE

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WHILST ulceration of the small intestine may occur in the course of diseases such as tuberculosis, Crohn's disease, typhoid fever, necrotizing enteritis, and the dysenteries, the occurrence of a solitary ulcer or, occasionally, multiple ulcers of the jejunum or ileum is sufficiently uncommon to arouse interest.

Ulcers associated with Meckel's diverticula, gastric heterotopia, and gastrojejunostomy are not considered here as they are secondary to a known aetiological factor.

Simple ulcers of the small intestine were first described by Matthew Baillie in 1805 and Cruivellier, discussing peptic ulcers in 1830, referred to 'simple ulcers' of the small intestine which he considered to be peptic in nature.

There have been few references to this condition in the British literature, but several American and Continental series of collected cases have been reported, the more outstanding of these being that of Brown and Pemberton (1936) and of Oudard and Jean (1925), since which time further reports and a comprehensive review by Ebeling (1933) have appeared.

Three further cases are here reported and the condition is briefly discussed.

Occurrence.—This condition affects all ages from 8 to 77 years and collected series indicate a preponderance of 3 males to 1 female.

Symptomatology.—Presentation may be of an acute nature, such as perforation, bleeding, or intestinal obstruction. The majority of cases present as perforation of a hollow viscus, and the diagnosis of perforated peptic ulcer is usually made preoperatively.

However, if the history is taken, it will be found that a high proportion of cases have had symptoms ranging from several weeks to three or four years before the onset of the acute episode. Some patients suffered from a dyspepsia strikingly similar to that of peptic ulceration in its relationship to meals and relief by alkalis. Yet others experienced bleeding presenting as either hæmatemesis or melæna. Some had undergone appendicectomy and many had been subjected to careful clinical and radiological examinations with no positive result.

One patient who had experienced repeated hæmorrhages was diagnosed to be suffering from 'Banti's syndrome' on account of splenomegaly consequent upon severe secondary anæmia.

As with peptic ulceration, stenosis may occur and with the onset of this state of affairs the dyspepsia has in several patients been superseded by the symptoms of progressive intestinal obstruction, as in Ravdin's case (1927). Fistulæ have not been recorded.

Pathology.—The ulcer or, rarely, ulcers are usually situated in one of two sites—the first loop of jejunum or the last loop of the ileum.

Macroscopically they present on the antimesenteric border of the small bowel and vary between 0·5 cm. and 2·5 cm. in diameter. The edge is terraced and the base may or may not be indurated. When perforated they show a striking similarity to the appearance of perforated duodenal ulcer, being clean and punched out.

Histologically the picture is that of an acute or chronic simple inflammatory reaction showing varying degrees of round-celled infiltration, necrosis, and fibrosis, together with thrombosis of the vessels. Chronicity predominates, which is in keeping with the long history given by most patients.

FIG. 779.—*Case* 2. Segment of ileum showing two separate perforations.

Annular constricting ulcers with an abundance of fibrous tissue are occasionally seen.

The lymph-nodes may be enlarged, and in these cases present a similar histological picture.

Simple ulcer of small bowel shows a marked tendency to perforation of all coats.

Causation.—Although there appear to be two distinct conditions, ileal ulceration and jejunal ulceration, the occasional occurrence of both forms in the same patient presumes a common aetiological factor.

The first and last loops of the small bowel are the most fixed, and consequently are more liable to suffer as a result of trauma and several cases have followed within some months of a definite abdominal injury.

Vascular causes such as thrombosis or embolism have been postulated. Focal infection has been suggested and some authors cite the work of Rosenow on the selective affinity of certain strains of streptococci for the digestive tract. Ulceration as a response to stress certainly does occur in the large intestine, but there is no evidence of a parallel association in the small intestine.

All of the postulated theories of causation have much experimental evidence to back their validity, but in the individual cases most authors conclude that the part played by any named aetiological factor is at best speculative.

However, the case quoted by Gale (1922) (*see below*) seems to be an exception.

Diagnosis.—The condition may present as an acute episode of perforation or intestinal obstruction and the diagnosis will be made at operation.

However, the problem of diagnosis of the chronic variety is of more than academic importance as it is in these cases that a multitude of investigations, and in many cases exploratory operations, are

performed, with failure to detect the causative disease process.

The problem of gastro-intestinal hæmorrhage, acute or chronic, in the absence of radiological evidence of disease of the œsophagus, stomach, duodenum, or colon, is a particularly common and perplexing condition which may from time to time be traced to simple ulcer of the small bowel. Occult blood in the stool is a frequent finding in the absence of obvious bleeding.

Ebeling (1933) points out that the condition may be diagnosed pre-operatively if careful radiological investigations are made, although barium studies of the small bowel are notoriously difficult of interpretation.

Treatment.—The acute episode, such as perforation and obstruction, will be treated surgically in any event, and in chronic cases where pre-operative diagnosis is made resection of the diseased segment of intestine is probably the safest treatment.

In acute perforations there is no place for wedge resection or oversewing of the ulcer, unless the patient's condition is critical.

The rare occurrence of carcinomatous ulcers of the small intestine makes resection, rather than a more limited procedure, desirable, as these ulcers may be impossible to differentiate at operation from the simple variety.

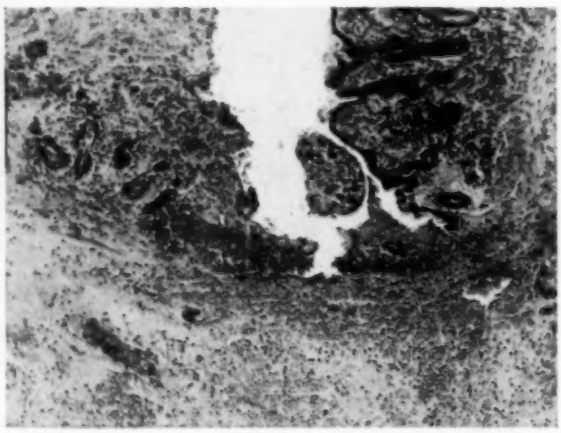

FIG. 780.—*Case* 1. Jejunal ulcer. The mucosa is thickened in the area surrounding the ulcer and there is an acute inflammatory reaction at the ulcer margin. The muscle is thickened by acute inflammatory œdema. Hæmatoxylin and eosin. (× 45.)

Prognosis.—The collected literature quotes a mortality of between 60 per cent and 75 per cent, and most of the deaths are due to peritonitis or hæmorrhage.

However, there is reason to suppose that these patients should fare no worse than perforated or bleeding ulcers of the stomach or duodenum.

The patient whose ulcer bleeds is at great risk, for he may be submitted to operation and the ulcer overlooked as it is in many cases not indurated and several reports are available of abdominal exploration with negative results being followed by demonstration of the bleeding ileal or jejunal ulcer at necropsy.

There is evidence to suggest that spontaneous healing does occur. Oudard and Jean (1925) reported the case of a patient who had a group of ileal ulcers in various stages of development, one of which was healing, and the stenotic type of lesion is probably an exaggerated response to the tissues to the ulcer.

Recurrence has been reported and Gale's patient had three separate episodes of perforation of the ileum, each of which followed an attack of furunculosis of the external auditory meatus.

CASE REPORTS

Case 1.—The patient was a young man aged 20 years, who was admitted as an emergency to Westminster Hospital under the care of Mr. Frank d'Abreu, on May 21, 1956.

There was no previous history of ill health or dyspepsia until the day of admission. Shortly after breakfast he experienced sudden upper abdominal pain and some minutes later vomited profusely.

ON EXAMINATION.—The patient showed the signs of perforation of a hollow viscus with acute generalized peritoneal irritation.

Investigations.—Plain radiography of the abdomen in the erect position revealed free air beneath both domes of the diaphragm. The vomit gave a positive reaction for occult blood.

A diagnosis of perforated peptic ulcer was made and surgical treatment advised.

AT OPERATION (Mr. John Mitchell).—Upper midline incision. On opening the peritoneum gas and blood-stained fluid escaped. A fibrinous exudate was seen to cover the upper loops of small bowel. There was no

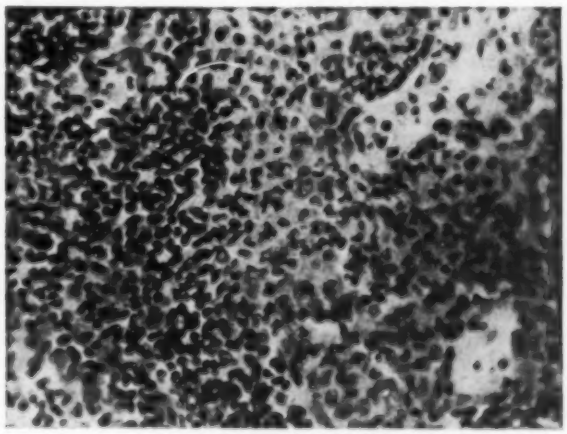

FIG. 781.—*Case* 1. Section of lymph-node. Acute inflammatory reaction showing infiltration with mononuclears and polymorphonuclear cells. Hæmatoxylin and eosin. (× 206.)

abnormality in the stomach or duodenum. Further inspection revealed a perforation 2 cm. in diameter situated 8 cm. from the duodenojejunal flexure on the antimesenteric border of the jejunum. One lymph-node in the adjacent mesentery was enlarged. No other abnormality was detected. The 8-cm. loop of jejunum bearing the perforation was resected together with the mesentery and enlarged node in relation to it and continuity restored by end-to-end anastomosis. The patient made a good recovery although this was delayed by post-operative adhesive obstruction which was relieved surgically.

PATHOLOGY REPORT.—The specimen consists of 8 cm. of jejunum bearing a perforated ulcer 2 cm. in diameter situated on the antimesenteric border. The mucosa was thickened in the area surrounding the ulcer. The mucous membrane is interrupted by an ulcer with an acute inflammatory reaction at the margin, and below this the muscle is thickened by acute inflammatory œdema

and exudate, the process extending right through to the peritoneum.

It is a non-specific type of reaction and the appearances are unlike those seen in peptic ulceration, Crohn's disease, or typhoid fever. The lymph-nodes are acutely inflamed, the œdema and infiltration with polymorphonuclear cells closely resembling the pathological changes present in the jejunum.

Case 2.—This patient was a man aged 55 years who developed a dyspepsia, post-prandial in nature and relieved by alkalis, together with colicky abdominal pain which recurred several times. The condition was investigated by barium meal and follow through, but no abnormality was detected.

One year later he developed a cough, and sputum and a radiograph of the chest revealed bilateral tuberculous infiltration. His sputum was positive for tubercle bacilli both on direct smear and culture.

He commenced a course of I.N.A.H. 50 mg. twice daily together with P.A.S. 15 g. daily and was admitted to the Brompton Hospital under the care of Dr. F. H. Young. Whilst an in-patient he was treated with a course of old tuberculin. He made a good clinical response to his treatment and by March, 1955, his sputum was no longer positive for tubercle bacilli, he gained weight, and his chest radiograph showed considerable clearing.

On the afternoon of April 17 he experienced sudden acute widespread abdominal pain.

On Examination.—The patient presented the picture of perforation of a peptic ulcer.

At Operation (G. Keen).—The peritoneal cavity was soiled with fæculent fluid, which was seen to originate from two punched-out perforations 0·5 cm. in diameter 20 cm. apart on the antimesenteric border of the ileum 2 m. from the ileocæcal valve. There were no adhesions and no lymphadenopathy. No other abnormality was found in the abdomen.

The loop of bowel bearing the perforations was resected and continuity was restored by end-to-end anastomosis.

The patient made a good recovery. Three months later he experienced an attack of colicky pain in the right iliac fossa which settled after three days, and when last seen in December, 1956, had had no further trouble.

Pathology.—The specimen is a segment of ileum 22 cm. in length showing four indurated areas of 1 cm. diameter on the antimesenteric border. Two of these are perforated and one is ulcerated on its mucosal surface.

Histologically a scattered diffuse inflammatory process characterized by round cells and lymphocytes has occurred in the submucous layer and there are present what appear to be occasional epithelioid cells. There is no caseation or other evidence of tuberculous infection.

Case 3.—This patient, a man aged 62 years, was admitted to The Brook General Hospital, Woolwich, on May 5, 1956, under the care of Dr. Bruce Pearson.

He complained of colicky abdominal pain and vomiting over the previous four days.

Past History.—The patient had been well until four years previously. He then developed paroxysms of abdominal pain, which were associated with constipation and abdominal distension. These attacks would last for a few days and then settle. Investigation suggested the possibility of a carcinoma of the splenic flexure of the colon and in August, 1952, laparotomy was performed. No abnormality was found. Since this time he experienced twelve further similar attacks from which he recovered spontaneously in most instances, but on several occasions was admitted to hospital. On one admission in July, 1953, he did not respond to conservative treatment and laparotomy was performed. This showed no abnormality other than a volvulus at the duodenojejunal flexure with no evidence of strangulation.

In December, 1953, he was readmitted with a further attack of severe abdominal pain and vomiting. On this occasion plain radiograph of the abdomen in the erect position showed free air to be present under the right dome of the diaphragm and a perforated peptic ulcer was thought to be responsible. This episode was treated by gastric suction and intravenous fluids and the patient made a good recovery. Several further admissions to hospital with acute abdominal symptoms followed. The case was further complicated by a known allergy to margarine and in 1954 he developed paralysis agitans.

On Examination.—The patient was pale and dehydrated. The abdomen was distended and bowel-sounds were exaggerated. He was treated once again by gastric suction and intravenous fluid therapy and made a satisfactory temporary recovery, but on May 25, 1956, vomited large amounts of altered blood. A transfusion of 3 pints of blood was given and he recovered, only to collapse on June 13 with a further large hæmatemesis. At this stage he developed bronchopneumonia and he died in coma on June 15.

Post-mortem Report.—All extra-abdominal organs, apart from the lungs, which showed bronchopneumonic changes, were normal.

Abdomen.—Three metres from the duodenojejunal flexure was a large transversally disposed ulcer 1·5 × 2·3 cm. in size. It was associated with stricture formation and the base was necrotic and hæmorrhagic. There was gross dilatation of the bowel proximal and collapse of the bowel distal to the ulcer. There were no other areas of ulceration present and no evidence of disease at the site of the previous volvulus at the duodenojejunal flexure.

DISCUSSION

Only *Case* 1 presented with perforation having had no previous symptoms.

Case 2 had symptoms over the previous twelve months and *Case* 3 was unique in that he at one time or another suffered from intestinal obstruction, perforation, and finally hæmorrhage, from which he died. Both of these cases had been fully investigated.

The occurrence of perforation of the ileum in *Case* 2 in a patient known to be suffering from pulmonary tuberculosis appeared at first sight to be a manifestation of tuberculous enteritis. However, the histology was unlike that of infection by the tubercle bacillus.

The ulcer of jejunum in *Case* 3 was well away from the site of the previous volvulus.

CONCLUSIONS

Simple ulcer of the small bowel is not a rare disease. Three cases are presented and a brief discussion of the literature of similar cases gives the impression that if the possibility of this condition is borne in mind, the correct diagnosis will be established more frequently.

Acknowledgements.—I am grateful to Mr. Frank d'Abreu for permission to publish *Case* 1; to Dr. Bruce Pearson for permission to publish *Case* 3; and to Dr. F. H. Young and Sir Russell Brock for permission to treat and report *Case* 2.

I also wish to thank Dr. Alan Morgan and Dr. Hallam for histological reports on *Cases* 1 and 2 respectively.

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SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF MULTIPLE CYSTS OF THE MESENTERY

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NUMEROUS cases of single mesenteric cyst have been recorded, but we have only been able to find 7 published cases of multiple mesenteric cysts in the literature of the last five years. The following case was therefore thought to be worthy of presentation.

FIG. 782.—Plain radiograph (erect) of the abdomen on admission, showing a few fluid levels in the small intestine, gas shadows in the colon (but not the rectum), and an absence of shadows in the right lower quadrant.

The additional interest of this case lies in the fact that the child was referred with an acute abdominal disorder, i.e., pain, distension, and rigidity, simulating acute peritonitis.

CASE REPORT

A boy aged 3 was seen in hospital at 8.0 p.m. The parents said that he seemed to have been in pain since 11.0 a.m. that day. The pain was situated around the umbilicus. He had been refusing his food, but there had been no vomiting, and the bowels had acted normally earlier in the day. The past history was negative.

ON EXAMINATION.—The child was lying quietly on a couch. Although pale, the temperature was 102° F., and the pulse 160. The tongue was slightly furred. The abdomen was moderately distended and rigid, more so on the right than on the left. There was generalized tenderness, and dullness to percussion in the right lower quadrant. On auscultation, the bowel sounds were diminished. On rectal examination, an impression was obtained of a highly placed soft mass on the right side. A plain radiograph with the patient erect (*Fig.* 782) showed the presence of a few fluid levels in the abdomen, probably in the small intestine. Gas was present in the colon. There was an absence of any gas shadows in the right iliac fossa.

DIAGNOSIS.—Acute peritonitis seemed the most likely diagnosis in view of the abdominal pain, distension, tenderness, reduced bowel sounds, and the radiographic evidence of small bowel dilatation. For a case of acute peritonitis, however, the degree of distension seemed to be out of proportion to the length of the history of pain. On the other hand, the mass palpated rectally felt like distended bowel suggesting intestinal obstruction. This, together with the area of dullness in the right iliac fossa and the absence of gas shadows in this region on X-ray examination, could have been due to an appendix mass or to a strangulated loop of bowel. Laparotomy was obviously required.

AT OPERATION.—Two large cysts were found in the mesocolon, one 17 cm. in diameter, and the other 8 cm. The sigmoid colon at this point was compressed and appeared to be partially obstructed. From the vicinity of these two large cysts, innumerable smaller ones extended upwards on to the posterior abdominal wall as high as the left kidney. The descending colon was unduly mobile and had a long mesentery; the sigmoid mesentery was considerably lengthened. As the mass of cysts was traced upwards, the individual cysts became smaller, their appearance simulating soap bubbles blown through a wire loop. The left kidney and ureter were normal. On further exploration, the only other abnormality to be found was enlargement of a number of lymph-glands in the mesentery of the *small* intestine. Simple excision of

all the abnormal tissue was obviously impossible owing to its adherence to the sigmoid colon and its vessels. The sigmoid colon was therefore resected, together with the two large cysts and several of the smaller ones (*Fig.* 783).

FIG. 783.—The specimen removed at operation, showing the two largest cysts, together with the portion of the sigmoid colon which the latter were compressing, and to which they were firmly attached.

An end-to-end anastomosis of the large bowel was performed.

After operation, the alimentary tract was decompressed by gastric suction. The child was maintained on intravenous therapy for 24 hours, after which time the temperature had fallen from 103° to 98°, and the pulse from 160 to 100. Bowel sounds were audible on the second day and a spontaneous bowel action occurred on the fifth day, and he made an uneventful recovery. When seen 9 months later there had been no further symptoms and the abdomen remained flat.

PATHOLOGY.—The specimen was reported on by Dr. P. M. Peters as follows: "A piece of child's large intestine 14 cm. long, partly enclosed by two large thin-walled cysts 17 and 8 cm. diameter respectively, and with clusters of smaller cysts up to 2·5 cm. diameter in the fatty mesentery. These smaller cysts contain water-clear fluid; the large ones were not opened but the fluid appears to be the same as seen through the translucent cyst walls. The lumen of the gut shows a saccular pouch opposite the attachment of each of the larger cysts, probably a traction diverticulum, but there is no communication with the cysts.

FIG. 784.—Microscopic section of part of the specimen, showing the cyst walls. H. and E. (× 56.)

"*Histology*: The section (*Fig.* 784) shows multiple lymphatic cysts of varying size, some of the larger ones having some smooth muscle in the wall. The appearances are very like those of cystic hygroma and probably represent a congenital cystic malformation of cystic lymphatic vasoformative tissue."

Table I.—DETAILS OF 8 CASES OF MULTIPLE MESENTERIC CYSTS

AUTHOR	AGE	SEX	MESENTERIC SITE	PRESENTATION	REPORTED PATHOLOGY	FLUID IN CYSTS	ASSOCIATED ABNORMALITY	TREATMENT
Present case	3 yr.	M.	Sigmoid	Acute peritonitis	Lymphatic cysts	Serous	Long sigmoid mesentery, together with presence of descending meso-colon	Resection of bowel
Kron and Satinsky (1954)	6 yr.	M.	Jejunum	Intestinal obstruction	No lining made out	Chylous	Cæcum in right upper quadrant	Enucleation of the cysts
Beyer and others (1953)	2½ yr.	M.	Ileum	Ascites	No report	Chylous	None	Radio-therapy
Burnett and others (1950)	28 yr.	F.	Sigmoid	Incidental finding at operation	No report	Serous	None	Enucleation
Standeven (1953)	31 yr.	F.	Ileum	Acute intestinal obstruction	Lymphangioma	Serous	Volvulus of ileum (? a complication of the cysts)	Enucleation
Lowman and others (1952) *Case* 1	16 yr.	M.	Ileum	Chronic abdominal pain	No lining made out	Chylous	Dilated lacteals	Resection of bowel
Case 2	16 mth.	M.	Ileum	Acute intestinal obstruction	No lining made out	Serous	Cæcum in left upper quadrant	Enucleation
Valke (1952)	2½ mth.	M.	Ileum	Acute intestinal obstruction	Cysts with endo-thelial lining	Chylous	Enormous lacteals	Resection of bowel

DISCUSSION

Mesenteric cysts have been reported as occurring in approximately 2 out of 200,000 patients attending hospital (Collins and Berdez, 1934), and 3 in 750,000 hospital admissions (Vaughn, Lees, and Henry, 1948). The incidence of multiple mesenteric cysts is very much lower than that of single cysts (Warfield, 1932). The 7 cases of multiple cysts reported during the last five years, together with our case, are analysed in *Table I*.

In cases of single mesenteric cyst, associated abnormalities are rare. Of the cases of multiple mesenteric cysts reviewed in *Table I*, *Cases* 6 and 8 were associated with dilated lymphatics, and *Cases* 2 and 7 had errors of intestinal rotation; in our case, the descending colon possessed a mesentery and the sigmoid mesentery was unduly long. In our opinion, this frequency of associated anomalies cannot be ignored; furthermore, it is felt that it may provide a clue to the aetiology of the condition in some cases. Nixon (1956 a) has produced evidence that in the young embryo, as the intestines fall back into the abdomen, a *temporary* volvulus may occur and lead to the development of intestinal atresia by interfering with the blood-supply to the bowel; he also states (Nixon, 1956 b) that cysts in the mesentery are not infrequently seen during operations for volvulus neonatorum. We therefore suggest that multiple mesenteric cysts may in some cases be due to an intra-uterine 'accident' to the lymphatics.

We are indebted to Mr. W. B. Gabriel, under whose care the child was admitted, for permission to treat the patient and to publish this report. Our thanks are also due to Dr. P. M. Peters for supplying the photographs of the specimen.

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MALIGNANT CHANGE IN A DENTIGEROUS CYST

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INTRODUCTION

MALIGNANT change in dental and dentigerous cysts is very rare and many of the cases reported as such seem to be cysts fortuitously associated with malignant disease of the jaw rather than malignant changes in a dental or dentigerous cyst. Darlington, Ehrlich, and Seldin (1953) consider their case to represent a carcinoma in a pre-existing dental cyst, and not an invasion from the antrum. They show no radiographs of the case, but report no recurrence two

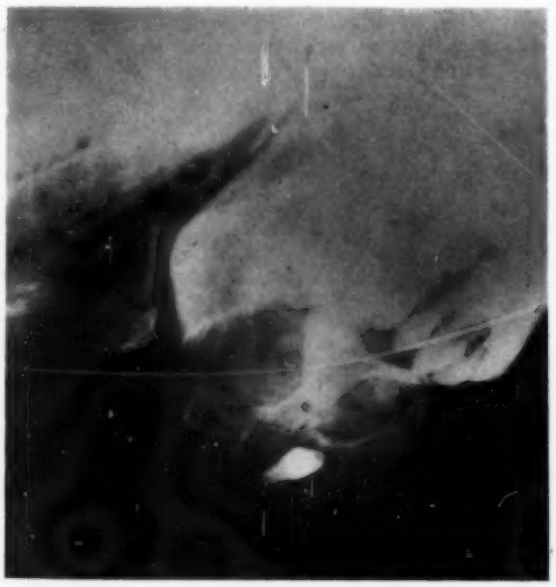

FIG. 785.—Lateral oblique radiograph of left mandible, showing gross expansion of the bone, and the premolar tooth contained in the cyst.

FIG. 786.—Occlusal radiograph of the jaw, showing the extension of the cyst across the midline.

years after resecting the maxilla. Sealey's (1947) case of a malignant dental cyst of the maxilla which fungated through the alveolus brings in the question of whether the carcinoma arose in the cyst or invaded the cyst from the alveolus. There is no follow-up reported, the tumour appeared to be very well differentiated and slow growing. In Frankl and Wiesner's (1949) case malignant change is reported in the maxilla following removal of a dental cyst six years previously. It is not possible in this case to ascertain from where the carcinoma arose. Thoma (1934) reports 2 cases; one is suggestive of cystic degeneration in an established carcinoma of the mandible, the other shows a dental cyst with a carcinoma extending into the surrounding mandible. Again the question arises of whether the carcinoma arose from the cyst or from outside the cyst and invaded it during its growth.

FIG. 787.—Lateral oblique radiograph of the left mandible 18 months post-operatively, showing consolidation and regeneration of the bone.

FIG. 788.—Occlusal radiograph of the jaw 18 months post-operatively for comparison with *Fig.* 786.

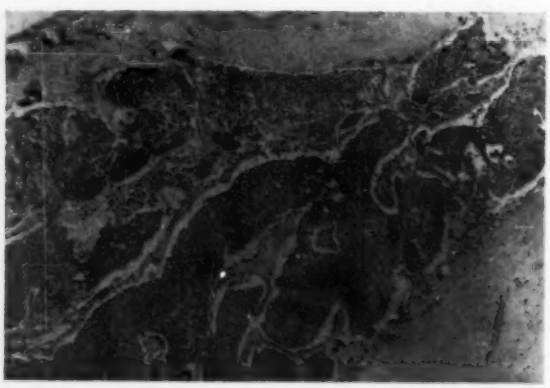

FIG. 789.—Photomicrograph of a section through the papilliferous part of the tumour. H. and E. (×50.)

CASE REPORT

A woman, aged 33 years, attended hospital complaining of a painless swelling of the chin and displacement of the lower teeth. The swelling had been present for two years, and had gradually increased in size.

ON EXAMINATION.—There was a smooth bony hard swelling extending from just distal to the mental foramen on the left side of the mandible across the midline to the canine region on the right side. Egg-shell crackling could be elicited on pressure. The swelling was not tender on palpation, nor attached to the skin. There were no palpable lymph-nodes, and no sensory nerve involvement.

Intra-oral examination showed the dental condition to be poor, $\overline{764|7}$ only being present. The remaining tooth on the left side, a second molar, was very loose and displaced lingually. The swelling noted on extra-oral examination extended from the lower second molar to the midline and obliterated the buccal sulcus.

Radiographic examination showed a large radio-lucent area in the mandible with an unerupted premolar tooth lying close to the lower border on the left side. The area extended from the ascending ramus on the left side, across the midline, to the first premolar on the right side. There was considerable expansion of the bone with thinning of the cortex (*Figs.* 785, 786). A radiographic diagnosis of follicular (dentigerous) cyst was made.

AT OPERATION.—The cyst was removed under general anæsthesia through a submandibular approach. The bone on the outer and inferior aspects of the cyst was very thin and easily removed, the mental nerve was identified and preserved, and the cyst enucleated. The bony cavity, which extended from the ascending ramus on the left side to the mental foramen on the right, was saucerized to eliminate 'dead space'. The lower left second molar and the lower right first premolar were denuded of bone and were removed, and the sockets closed tightly with black silk sutures. The submandibular incision was closed in layers incorporating polythene tubing for continuous low pressure suction drainage. The drainage tube was removed after 48 hours and the skin sutures after 5 days. The wound healed without trouble.

The patient has been followed up both clinically and radiographically and 18 months post-operatively there is no evidence of recurrence (*Figs.* 787, 788).

PATHOLOGICAL REPORT.—The collapsed cyst, together with an adjacent submandibular lymph-gland and two pieces of overlying bone, was submitted for histological examination. The collapsed cyst was in two pieces,

measuring 55 × 25 × 25 mm. and 22 × 14 × 14 mm. The outer surface of the cyst wall presented a nodular appearance and the wall varied in thickness from 1 to 5 mm. Some of the thicker areas on the inner surface of the wall were covered by papilliferous tissue. Histological examination of the thickened areas of the cyst wall showed squamous-cell carcinoma in the form of a papillary mass invading the wall (*Fig.* 789). No squamous epithelium of the type usually found in dental or dentigerous cysts could be found; it was not possible therefore to demonstrate a transition from the simple cyst epithelium to squamous-cell carcinoma shown in *Fig.* 789 The tumour did not appear to invade any of the body fragments submitted for examination. The lymph-node was free from invasion.

DISCUSSION

A case is described of what initially appeared to be a simple dentigerous cyst, but on microscopical examination showed undoubted squamous carcinoma in the wall. This appears to be unique since no other case reported has contained a tooth in the cyst.

A dentigerous or follicular cyst is one that develops around the crown of an unerupted tooth after the enamel has been formed. The epithelium of the tooth follicle proliferates, breaks down, and liquefies, and the cyst expands and resorbs the surrounding bone. There is no authoritative statement on the speed of development or growth. It is probable that in the case of dentigerous cysts the cyst originates soon after the enamel has been completed. In the case described this would mean that the cyst has been developing for at least twenty-five years. This is evidence against the cyst having originally contained malignant cells.

In view of the malignancy of the cyst a decision had to be made as to whether any further surgical treatment was advisable. It was thought preferable to reserve deep X-ray treatment for use if there was a recurrence of the tumour. The extent of the cyst, especially its extension across the midline involving the anterior attachment of the tongue, made resection of the affected part of the mandible a hazardous procedure. The fact that the cyst had enucleated easily from the bone and that the tumour seemed to be a papilliferous growth on the inner aspect of the cyst, was felt to justify no further operative treatment at that time.

Acknowledgements.—We would like to thank Mr. R. H. Boggon for permission to publish this case.

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GIANT-CELL GRANULOMATOUS THICKENING OF THE GASTRIC PYLORUS OF PROBABLE SARCOID ORIGIN

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ENOUGH cases have been published to establish diffuse thickening of the pyloric region of the stomach by a giant-cell granuloma as an entity. Some 5 cases have been traced in the literature (Gore and McCarthy, 1944; Orie, Van Ryssel, and Van der Zwaag, 1950; Appell, Prtizker, and Klotz, 1951; McKusick, 1953; and Allen, Batten, and Jefferson, 1956). The condition, however, still presents a problem in diagnosis and histogenesis. Most cases are diagnosed as carcinoma of stomach. Any condition, however rare, which mimics carcinoma of the stomach and carries a better prognosis is important. For these reasons a further case of giant-cell granuloma of the pylorus of the stomach is described and its histogenesis discussed.

CASE REPORT

A man, aged 38, was admitted on Jan. 29, 1957, complaining of upper abdominal discomfort for about four months. The pain, moderately severe, came on almost at once after meals and disappeared spontaneously a few hours later. For the last few weeks the pain had also gone through to the back between the scapulæ. He had lost 3 st. in weight over the past four months. There had been no nausea or vomiting. His only serious illness in the past was removal of a gangrenous appendix in July, 1950.

ON EXAMINATION.—He was a healthy man and no abnormal physical signs were elicited. A barium meal was carried out and the report was: "No œsophageal lesion was demonstrated. Stomach: the whole of the pyloric antrum region was rigid and there appeared to be an extensive infiltration of gastric wall here, the appearances being consistent with a linitis plastica. The duodenal cap appeared normal".

AT OPERATION.—Because of this X-ray report and clinical history a laparotomy was performed on Feb. 4 (Mr. P. H. R. Ghey). The abdomen was opened through a right paramedian incision. There was diffuse thickening of the pyloric region but no definite tumour could be felt. The lymph-glands were not enlarged and the rest of the abdomen appeared normal. A Billroth I partial gastrectomy was performed. The post-operative course was uneventful. The patient was discharged on Feb. 15 and has been followed-up in the Out-patient Department, where he has always been extremely well and symptom-free.

INVESTIGATIONS, some of which were performed after the receipt of the report on the histology of the stomach.—
Wassermann and Kahn reaction negative.
Serum-proteins 6·6 mg. per 100 ml. with a normal electrophoretic curve.
Occult blood in fæces negative.
Hæmoglobin 14·2 g. per 100 ml.
Leucocyte count 4700 per c.mm.
Blood-urea 32 mg. per cent.
Radiograph of chest hands, and feet: "No abnormality and no evidence of sarcoidosis".
Mantoux 1–1000 negative.

HISTOLOGY.—Macroscopically in the portion of stomach removed (lesser curvature 8 cm., greater curvature 16 cm.) the normal rugal pattern in the distal half

FIG. 790.—An illustration of the barium meal, showing rigidity of the pyloric region.

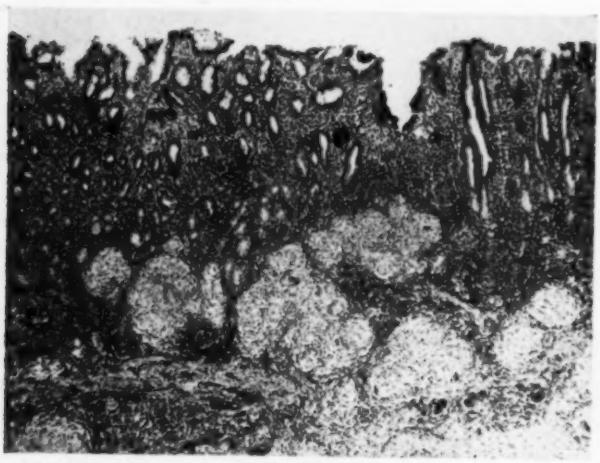

FIG. 792.—Gastric mucosa infiltrated with numerous sharply demarcated follicles. H. and E. (× 30.)

was completely replaced by numerous nodules of up to 1 cm. in diameter (*Fig.* 791). The lymph-nodes on the lesser and greater curvature measured up to 1 cm. in length.

Microscopically in the nodular area the stomach wall contained numerous clear-cut follicles composed of epithelioid cells, a few lymphocytes, and some giant cells, with up to 50 peripherally placed nuclei and a very occasional asteroid body. The follicles, although present throughout the stomach wall, were maximal in the mucosa just above the muscularis mucosæ (*Fig.* 792). The lymph-nodes also contained scattered follicles (*Fig.* 793).

DISCUSSION

A case of diffuse thickening of the pylorus of the stomach by a giant-cell granuloma has been described. The clinical picture was that of peptic ulceration; in contrast the 5 other reported cases all presented as pyloric stenosis. Our case, after a barium meal, showed pyloric thickening which was diagnosed as a

FIG. 791.—Partial gastrectomy specimen with nodular appearance of the mucosa in the distal half.

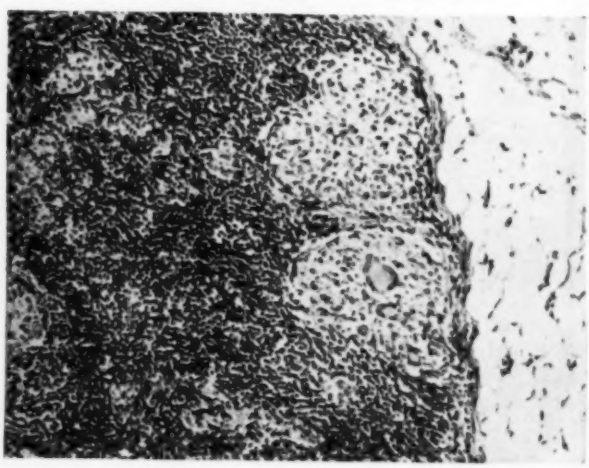

FIG. 793.—Follicles containing giant cells in a lymph-node. H. and E. (× 90.)

carcinoma. Even when reviewed with the correct diagnosis established, the appearances of the pylorus could not be distinguished from those of a carcinoma. The 5 other cases were also reported as carcinoma after X-ray examination. Thus it would appear that radiology of the stomach alone is by no means a certain diagnostic test, unless as in the case described

by Allen, Batten, and Jefferson (1956), the duodenum is also affected. Help may also be given in diagnosis by the involvement of other organs by the granulomatous process, as in the case of Orie, Van Ryssel, and Van der Zwaag (1950), which showed fibrosis of the lungs.

The cause of a giant-cell granuloma in a single organ is often difficult to determine; in our case tuberculosis could probably be excluded by the absence of caseation, the lack of involvement of the chest, and by a negative Mantoux reaction and syphilis by the negative Wassermann reaction. In our opinion the structure of the follicles, namely their clear-cut edge, the giant cells with a very occasional asteroid body, and the predominance of epithelioid cells, clearly suggest sarcoidosis as the cause. This opinion is in agreement with the authors of the other five reported cases.

However, we cannot entirely exclude Crohn's disease in our case and this is so in any case where the lesions are limited to the alimentary canal. Crohn's disease of the stomach has been reported by Martin and Carr (1953) but the terminal ileum was involved as well. The terminal ileum is nearly always involved in Crohn's disease and lack of this feature in our case is a strong point against Crohn's disease. Furthermore Crohn's disease does not usually produce the clear-cut follicles with numerous giant cells present in our case. The involvement of other organs, the lung in the case of Orie and others (1950) and the liver in the case of Allen and others (1956) strongly suggests that cases of diffuse thickening of the pylorus of the stomach by a giant-cell granulomatous reaction are due to sarcoidosis rather than Crohn's disease.

SUMMARY

A case of giant-cell granulomatous thickening of the pylorus is described. Enough cases have been published to establish this condition as an entity. The clinical features may be those of peptic ulceration or pyloric stenosis. The histogenesis is probably sarcoid in origin.

We wish to thank Dr. L. Cole and Mr. P. H. R. Ghey for permission to publish the clinical details; Dr. A. M. Barrett and Mr. V. C. Pennell for their help in the preparation of this paper; Dr. D. Gregg for the radiology report; and Mr. E. W. Mitchell and Mr. S. W. Patman for the illustrations.

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REVIEWS AND NOTICES OF BOOKS

A Manual of Human Anatomy. In five volumes. Vols. I–IV: By J. T. AITKEN, M.D., Reader in Anatomy at University College, London; G. CAUSEY, M.B., F.R.C.S., Professor of Anatomy, Royal College of Surgeons, London; J. JOSEPH, M.D., M.R.C.O.G., Reader in Anatomy, Guy's Hospital, London; and J. Z. YOUNG, M.A., F.R.S., Professor of Anatomy, University College, London. Vol. V: By J. T. AITKEN; D. A. SHOLL, B.Sc., Reader in Anatomy, University College, London; and J. Z. YOUNG. 8½ × 5½ in. Vol. I, Thorax and Upper Limb. Pp. 162 + viii, with 36 illustrations. 14s. Vol. II, Head and Neck. Pp. 180 + viii, with 53 illustrations. 16s. Vol. III, Abdomen and Pelvis. Pp. 135 + viii, with 29 illustrations. 12s. 6d. Vol. IV, Lower Limb. Pp. 117 + viii, with 36 illustrations. 12s. 6d. Vol. V, Central Nervous System. Pp. 140 + viii, with 40 illustrations. 1956–7. Edinburgh: E. & S. Livingstone Ltd. 12s. 6d.

FROM time to time dissecting manuals have appeared as alternatives to "little Cunningham", which has been such a trustworthy guide to so many members of our profession. The most recent rival is the work of four London teachers of anatomy and is in five volumes. These are well printed in clear type and illustrated by line drawings, some of which are in colour. The spiral binding adds to their usefulness and their plastic coloured covers to their attractiveness. Instructions for practical classes in osteology and surface anatomy conclude each of the first four volumes, while the fifth, that on the nervous system, deals also with a certain amount of microscopical anatomy. The five volumes deal in order with the thorax and upper limb, the head and neck, the abdomen and pelvis, the lower limb, and the central nervous system, a new order of dissection for those of us who, following Cunningham, began our dissecting with the upper part of the back followed by the upper limb. In each volume of the new book an attempt is made to link together development, structure, and function with the applications to clinical practice. Much of the detail found in dissecting manuals is absent and there are paragraphs indicating the functional aspects of the parts being dissected, an unusual feature of such books. This may be helpful to the immature undergraduate for whom the work is obviously intended. It is the authors' expressed intention that the book should be a guide to what is required of the student to pass his second medical examination, but opinions will differ as to whether it is sufficiently detailed for this, having regard to the fact that nowhere is he referred to a reliable standard book on anatomy from which to obtain more information to build on these rather bare foundations.

Certain errors are apparent, e.g., on page 126, Vol. I, the ulnar nerve is stated to pass through the flexor retinaculum into the palm; on page 8 of Vol. II, longissimus capitis is stated to be deep to semispinalis capitis; and in Fig. 12, Vol. IV, page 35, the insertion of the semimembranosus is incorrectly depicted. The brief reference to hernia does not do justice to this important condition which the student should thoroughly understand.

Various omissions are to be expected in a condensed account of regional anatomy, but as constituents of the

spermatic cord, the lymphatics and nerves of the testis and the cremasteric artery should be mentioned.

As a guide to dissection this book is open to some criticism when compared with the for so long unrivalled and so widely used Cunningham's *Manual of Practical Anatomy*. By being too brief and too matter-of-fact it fails to set a sufficiently high standard. Not the least of the attractions of Cunningham and one which has delighted generations of students, is the style in which it is written, e.g., "By their simultaneous contraction they (the transverse perinei) steady the central point of the perineum. . . . But what is the central point of the perineum? It is . . .", etc. Nothing comparable is to be found in the present manual, but when we have said this we must admit our admiration for the enterprise of its authors and their admirable aim of associating development, structure, and function in such a way as to lessen the student's burden. The book must be regarded as an experiment, the result of which will be watched with interest, but should this new approach to dissection become popular with teachers and students, some expansion as well as revision and correction will be desirable in future editions.

Lumbar Puncture and Spinal Analgesia. By Sir ROBERT MACINTOSH, M.A., D.M., F.R.C.S.(Edin.), F.F.A.R.C.S., Nuffield Professor of Anæsthetics at University of Oxford. Second edition. 8¾ × 6 in. Pp. 142 + viii, with 110 illustrations. 1957. Edinburgh: E. & S. Livingstone Ltd. 25s.

THE second edition of this admirable little book contains a number of minor alterations in the script, and the illustrations have been revised. All Sir Robert Macintosh's books are well known for their excellent and profuse illustrations, and this is no exception. Many illustrations are in colour, and those who read this book carefully will end with an excellent knowledge of the anatomy of the spinal canal. The chapters are all clearly written and each chapter of the total nine is filled with sound information. This is a book which should be in the library of every anæsthetist and those interested in the technique of lumbar puncture.

Surgery of Head and Neck Tumors. By HAYES MARTIN, M.D., Attending Surgeon, Memorial Hospital, Associate Professor of Clinical Surgery, Cornell University Medical College, New York. 10½ × 7½ in. Pp. 430+xvi, with 599 illustrations. 1958. London: Cassell and Co. Ltd. £6 17s. 6d.

To British readers the title of this book is misleading. It is, in fact, a manual of operative management and technique. It does not include skull or intracranial tumours, but operations for a few conditions which are not tumours, e.g., thyroglossal and branchial cysts and fistulæ and œsophageal diverticula, are described. All alternative methods of the management of malignant tumours are excluded and there is no discussion about the indications for operative treatment as compared with radiotherapeutic methods. In fact, the impression is given that there is no alternative to surgical ablation.

The first quarter of the book deals with the general management of operations for tumours in this region while the remainder is an atlas of operative technique illustrated by many clear line drawings. On this aspect the surgeon will find all he wants to know and there are points of detail which will be valuable to any surgeon who has to treat malignant disease of the mouth and neck.

The author is known for his radical methods, particularly in the combined operations for removal of the primary tumour and a block dissection of the lymph drainage in one piece. These operations are well described. However, some of the very limited operations for cancer, such as the local excision of a tumour of the tongue, described on pages 266 and 267, should have no place in modern therapy. There is also no mention of the valuable use of the drill and Gigli saw in the partial resection of the body of the mandible. Surely the removal of part of the alveolus with rongeur forceps after a cancer of the overlying gum has been excised as a separate piece, as shown on page 226, is contrary to the principles of present-day practice?

To sum up, this book will provide some useful information regarding technique for the surgeon who already has mature judgement, but it may well prove to be dangerous in the hands of the less experienced. In no sense can it be regarded as a guide to the management of tumours in this area, for it deals with one method of treatment only.

Cleft Palate and Speech. By MURIEL E. MORLEY, M.Sc., F.C.S.T., Speech Therapist-in-Charge of the Speech Therapy Unit, the United Newcastle upon Tyne Teaching Hospitals. Fourth edition. 7¼ × 4¾ in. Pp. 271 + xx, with 86 illustrations. 1958. Edinburgh and London: E. & S. Livingstone Ltd. 27s. 6d.

EVERY surgeon who operates upon cleft-palate cases knows how much the end-result of his efforts are due to the constant help of the trained speech-therapist.

Miss Morley is now publishing the fourth edition of her book. Four editions in eight years speaks for itself and stamps the book not only as a useful one but one that is in constant demand.

Mr. Richard Battle wrote the Foreword to the first edition and has no hesitation in recommending it. Professor T. Pomfret Kilner wrote the Foreword to the second edition and was loud in his praise for it. Since then this book requires no Foreword for it is known not only in this country but also overseas.

As each new edition comes out Miss Morley has something new to teach us, and in this edition the further experience of the author is given, together with an account of the follow-up of cleft-palate cases.

When it is considered that the incidence of cleft-palate cases is 1·5 per 1000 births, or 1 in every 665 live births, there must be a large number of cleft-palate cases to be dealt with.

Once again we have nothing but praise for Miss Morley's useful book.

Principles of Surgical Management. By H. A. F. DUDLEY, F.R.C.S.E., Lecturer in Surgery, University of Edinburgh; B. C. PATON, M.R.C.P.E., The Royal Infirmary, Edinburgh; I. F. MACLAREN, F.R.C.S.E., The Royal Hospital for Sick Children, Edinburgh; I. B. TAIT, F.R.C.S.E., St. Mary's Hospital, London; and G. F. MURNAGHAN, F.R.C.S., F.R.C.S.E., The Western General Hospital, Edinburgh. With a Foreword by JOHN BRUCE, C.B.E., T.D., F.R.C.S.E., Regius Professor of Clinical Surgery, University of Edinburgh. 10 × 7 in. Pp. 203 + viii, with 68 illustrations. 1958. Edinburgh and London: E. & S. Livingstone Ltd. 27s. 6d.

HERE is a new practical book which should interest all surgeons. This book sets out in a most lucid way the many and varied methods now in use in the after-care of surgical cases. It is illustrated with a large number of original drawings, all of which are well chosen. There are many practical surgical tips which should prove of real value to the house surgeon or registrar who requires to keep rubber tubes in a particular place.

At the end of the book there is a useful appendix giving the amounts of blood required for analysis and normal values of common biochemical determinations.

A most useful appendix for ready and easy reference. So many of these values cannot possibly be remembered, and some sort of table such as this is very necessary and useful.

A table of drugs used in the after-treatment of surgical cases is given together with their proprietary equivalent.

A most useful book which can be recommended wholeheartedly.

Frazer's Anatomy of the Human Skeleton. Edited by A. S. BREATHNACH, M.D., M.Sc., Senior Lecturer in Anatomy, St. Mary's Hospital Medical School, London. Fifth edition. 11¼ × 7¾ in. Pp. 247+viii, with 197 illustrations, many in colour. 1958. London: J. & A. Churchill Ltd. 50s.

A NEW edition of 'Frazer' is an event, particularly as this follows the previous one after an interval of eighteen years, which is a longer period than that separating any previous editions. Nevertheless, the format of the book has been preserved, and the present edition has retained most of those delightful pen and colour-wash drawings so familiar to generation after generation of 'Primary' students, embellishments which give this classic work its character and constitute perhaps its main attraction. Such alterations and additions as have been made to the text have enhanced the value of the work and these are mainly concerned with the vertebral column and with the ribs and sternum. New material dealing with the movements of the shoulder girdle and of the semilunar cartilage of the knee has been included, which brings the account of these joints up to date.

It is generally admitted that instruction in anatomy should nowadays concern itself with the radiological appearances of living structures and so link this subject with clinical studies. To this end, therefore, a number of X-ray plates have been introduced. Excellent though the quality of these plates is, however, it is doubtful whether in a work of this nature they add very much of value.

'Frazer' has traditionally been a book for post-graduate study and the inclusion, for instance, of an X-ray picture of a normal elbow-joint or lumbar spine will not serve to instruct the readers of this book, to whom such photographs must be commonplace.

Once again, 'Frazer' will take its place on the shelves of all postgraduate students of anatomy and surgery. It has been written so obviously by a lover of his subject and contains descriptions of so many original and unusual aspects of topographical anatomy that it is a joy to read, and converts the task of acquiring hard facts into a stimulating adventure.

A Contribution to the Study of Portal Hypertension. By ALAN HENDERSON HUNT, M.A., D.M., M.Ch., F.R.C.S., Surgeon, St. Bartholomew's Hospital, and the Royal Marsden Hospital; Late Hunterian Professor, Royal College of Surgeons of England. 9¾ × 6¼ in. Pp. 230+xii, with 119 illustrations. 1958. Edinburgh and London: E. & S. Livingstone Ltd. 40s.

THE background to this excellent monograph was the Jacksonian Prize of the Royal College of Surgeons for the year 1956. The author has supplemented this with further clinical cases and has included in this book some 120 illustrations which enhance the written word considerably. The time was ripe for a practical yet comprehensive volume on the surgery of portal hypertension and Mr. Alan Hunt has supplied this in no uncertain terms.

This monograph is a credit to British surgery, and we would like to congratulate the author on an excellent production.

Cardiovascular Innervation. By G. A. G. MITCHELL, O.B.E., T.D., M.B., Ch.M., D.Sc., Professor of Anatomy and Director of the Anatomical Laboratories in the University of Manchester. With a Foreword by Sir GEOFFREY JEFFERSON. 9¾ × 6¾ in. Pp. 356 + xii, with 217 illustrations, many in colour. 1956. Edinburgh: E. & S. Livingstone Ltd. 55s.

IT is almost impossible to review this book except in general terms because it is packed full of so much, and excellent, detail. As Sir Geoffrey Jefferson states in the Foreword, "Works on the anatomy of the nerve supplies of the cardiovascular system are few indeed and correct accounts rarer still". The impression created by the masterly presentation and the profuse, beautiful, and clear illustrations is that here indeed the account is correct. The numerous illustrations, each such a pleasure and profit to look at, are a very notable feature.

Although it is primarily anatomical, this book contains much in addition to anatomy. Its author knows well that an account of cardiovascular innervation must include much that is physiological and clinical. This scholarly and convincing presentation of the subject is enhanced by the references, which indicate an immense labour.

In addition to the full description of the peripheral part of the autonomic nervous system, about one-third of the book deals with the central pathways and connexions. It is clear that the material covers a field so wide that it presents valuable information to those working in many branches of clinical medicine and surgery, as well as to the pure anatomist and physiologist.

The publishers have done an excellent job in their presentation of the material; paper, printing, and reproduction of illustrations are all pleasurable and perfect. It is difficult to speak too highly of such a book, which will certainly become a classic. It is recommended unreservedly.

Recent Advances in Urology. By HOWARD G. HANLEY, M.D., F.R.C.S., Surgeon, St. Peter's, St. Paul's, and St. Philip's Hospitals; Urologist, Hillingdon and Harefield Hospitals; Lecturer in Urology, University of London. With the assistance of specialist contributors. 8¼ × 5¼ in. Pp. 270 + viii, with 83 illustrations. 1957. London: J. & A. Churchill Ltd. 30s.

IT was high time that the subject of urology was represented in the *Recent Advances* series; the previous volume of 1936 was long out of date, and Mr. Hanley has succeeded in producing another which reflects the many changes in this rapidly progressing branch of surgery. He has enlisted the aid of five collaborators to discuss branches in which they have special knowledge. Urologists now recognize that their work is as much medical as surgical and it is appropriate that the first chapter, by Dr. A. R. Harrison, is devoted to Medical Problems in Urology, and the second, by Dr. K. F. Anderson, to the Chemotherapy of Urinary Infections. Mr. Chapman writes on Periurethral Prostatic Resection; the term 'Transurethral' is in more common usage although possibly less correct. Mr. J. D. Fergusson contributes a scholarly chapter on Prostatic Cancer, and Mr. D. M. Wallace gives the modern views on the aetiology, classification, and treatment of Bladder Tumours.

The major part is written by Mr. Hanley himself, and whilst his particular experience of the image amplifier makes his account of radiology of the genito-urinary tract of especial interest to urologists, the chapters on renal surgery will have the greatest appeal to general surgeons. To single out one where all are good is not easy, but we would commend the masterly account of genito-urinary tuberculosis to all who may be called upon to treat that disease. The changed ideas of recent years are not as widely known even now as they should be; bacteriological

diagnosis is of more importance than radiological if early treatment is to be instituted and this side of the problem has not kept pace with modern therapy. The principles of medical treatment are well described and the indications for surgery clearly given, stress being laid on the scope for conservative surgery. That the need for sanatorium treatment is as great in renal as in pulmonary tuberculosis can no longer be denied, and this account should do much to convince those in authority that such accommodation must be provided.

The book is well produced, the illustrations are clear, and the bibliography is well chosen. A reasonable price has been made possible by the moderate size of the volume.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest to our readers.*]

Transactions of the International Society of Plastic Surgeons. First Congress, Stockholm and Uppsala. 1955. Edited by TORD SKOOG, M.D., General Secretary of the Congress, in co-operation with ROBERT H. IVY, M.D., and the Editorial Board of Plastic and Reconstructive Surgery. 10 × 6½ in. Pp. 568 + xvi, with 467 illustrations. 1957. Baltimore: Williams & Wilkins Co. (London: Baillière, Tindall & Cox). 160s.

Cardiovascular Collapse in the Operating Theatre. By HERBERT E. NATOG, M.D., Assistant in Anæsthesia, University of Illinois, College of Medicine; and MAX S. SADOVE, M.D., Professor of Surgery (Anæsthesiology), University of Illinois College of Medicine. With a Foreword by WARREN H. COLE, M.D. Pp. 197 + xvi. 1958. London: Pitman Medical Publishing Co. Ltd. 45s.

Ulcerative Colitis. By HARRY E. BACON, B.S., M.D., Sc.D., Ll.D., F.A.C.S., F.A.P.S., Professor and Head of Department of Proctology, Temple University Medical Center, Philadelphia, Pa. With a Foreword by ALTON OCHSNER, B.A., M.D., Sc.D., F.A.C.S., F.A.P.S., Professor of Surgery, Tulane University School of Medicine; Director of Surgery, Ochsner Clinic and Ochsner Foundation Hospital, New Orleans. 10 × 6¾ in. Pp. 395 + xx, with 183 illustrations. 1958. Philadelphia: J. B. Lippincott Co. (London: Pitman Medical Publishing Co. Ltd.). 120s.

Surgery in Infancy and Childhood. By MATTHEW WHITE, M.A., M.B., Ch.B., F.R.F.P.S. (Glas.), F.R.C.S. (Edin.), Consulting Surgeon, Royal Hospital for Sick Children, Glasgow; and WALLACE M. DENNISON, M.D., F.R.F.P.S. (Glas.), F.R.C.S. (Edin.), F.I.C.S., Barclay Lecturer in Surgery in Relation to Infancy and Childhood, University of Glasgow; Surgeon, Royal Hospital for Sick Children, Glasgow. 8¾ × 5¾ in. Pp. 444 + xii, with 266 illustrations. 1958. Edinburgh and London: E. & S. Livingstone Ltd. 45s.

Tumors of the Soft Somatic Tissues. A Clinical Treatise. By GEORGE T. PACK, M.D., Ll.D., F.A.C.S., Attending Surgeon, Memorial Center for Cancer and Allied Diseases; Associate Professor of Clinical Surgery, Cornell University Medical College; and IRVING M. ARIEL, M.D., F.A.C.S., Associate Clinical Professor of Surgery and Associate Attending Surgeon, New York Medical College. 10¼ × 6½ in. Pp. 820 + xviii, with 652 illustrations. 1958. London: Cassell & Co. Ltd. 11 gns.

Homosexuality Transvestism and Change of Sex. By EUGENE DE SAVITSCH, M.D. 7⅛ × 4½ in. Pp. 118 + viii, with 6 illustrations. 1958. London: William Heinemann Medical Books Ltd. 12s. 6d.

The Tissues of the Body. An Introduction to the Study of Anatomy. By W. E. LE GROS CLARK, F.R.S., Fellow of Hertford College, Dr. Lee's Professor of Anatomy in the University of Oxford. Fourth edition. 9¼ × 5¾ in. Pp. 415 + xii, with 127 illustrations. 1958. Oxford: Clarendon Press (Oxford University Press). 40s.

Reflexogenic Areas of the Cardiovascular System. By C. HEYMANS, M.D., Professor of Pharmacology, University of Ghent; and E. NEIL, M.D., D.Sc., John Astor Professor of Physiology, University of London, Middlesex Hospital Medical School. 9¾ × 7 in. Pp. 271 + viii, with 89 illustrations. 1958. London: J. & A. Churchill Ltd. 56s.

The Cerebrospinal Fluid. A Ciba Foundation Symposium. Edited by G. E. W. WOLSTENHOLME, O.B.E., M.A., M.B., B.Ch.; and CECILIA M. O'CONNOR, B.Sc. 8 × 5 in. Pp. 335 + xii. Fully illustrated. 1958. London: J. & A. Churchill Ltd. 50s.

Clinical Neuro-surgery. The Proceedings of the Congress of Neurological Surgeons, Chicago, Illinois. 1956. Volume 4. 9 × 5¾ in. Pp. 142 + x, fully illustrated. 1957. Baltimore: Williams & Wilkins Co. (London: Baillière, Tindall & Cox). 56s.

Microscopic Anatomy of the Temporal Bone. By DOROTHY WOLFF, A.B., M.A., Ph.D., Research Pathologist, Manhattan Eye, Ear and Throat Hospital; RICHARD J. BELLUCCI, B.S., M.S., M.D., Surgeon-in-Charge, Speech and Hearing Clinic, Manhattan Eye, Ear and Throat Hospital; and ANDREW A. EGGSTON, B.S., M.D., Pathologist and Director of Laboratories, Manhattan Eye, Ear and Throat Hospital. 9¼ × 7¼ in. Pp. 414 + x, with 199 illustrations. 1957. Baltimore: Williams and Wilkins Co. (London: Baillière, Tindall & Cox). 100s.

The Life and Work of Thomas Porter McMurray. By BRYAN MCFARLAND, Professor of Orthopædic Surgery, University of Liverpool. 8½ × 5½ in. Pp. 12. 1958. Liverpool: Liverpool University Press. 5s.

Sherrington: Physiologist, Philosopher and Poet. By LORD COHEN OF BIRKENHEAD, M.D., D.Sc., Ll.D., F.R.C.P., F.A.C.P., F.F.R., F.S.A., J.P., Professor of Medicine, University of Liverpool. 8½ × 5½ in. Pp. 108 + xiv, with 17 illustrations. 1958. Liverpool: Liverpool University Press. 17s. 6d.

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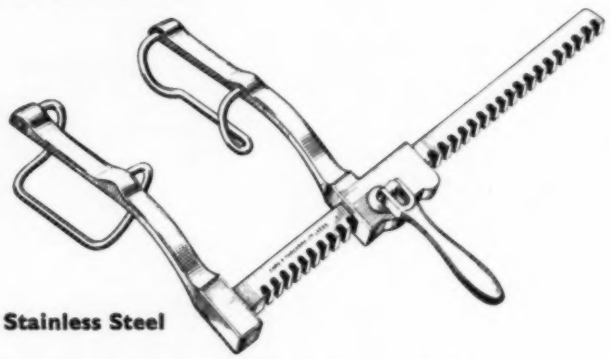

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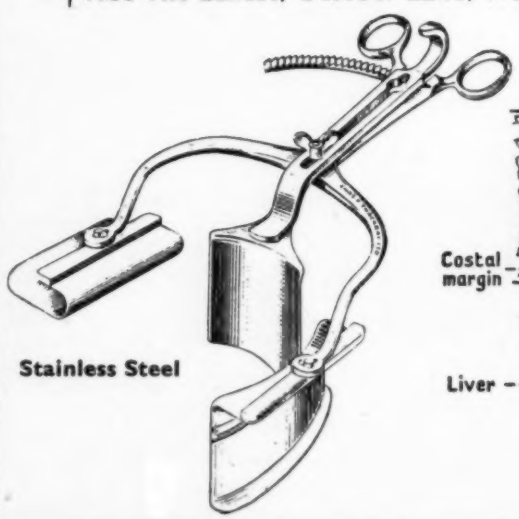

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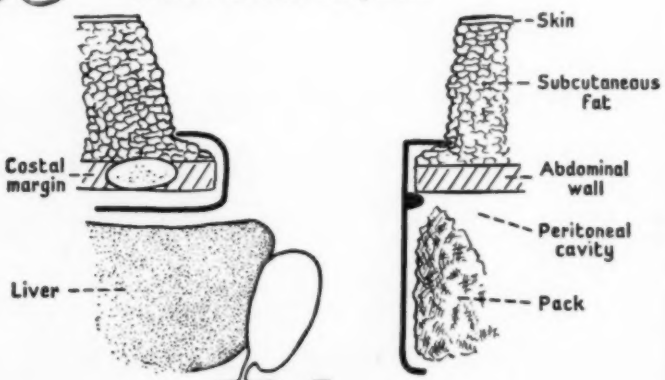

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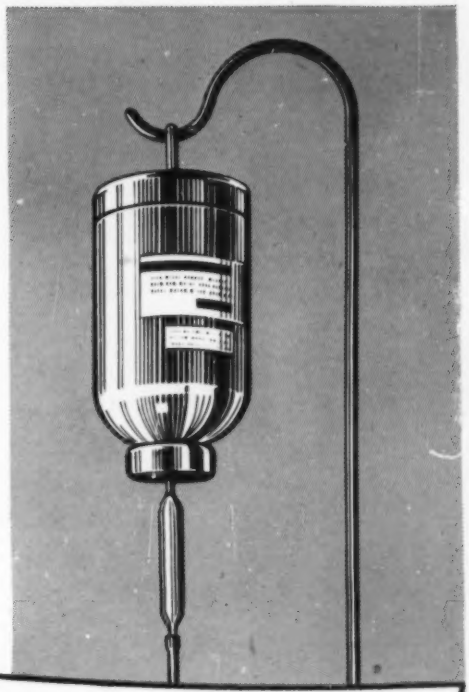

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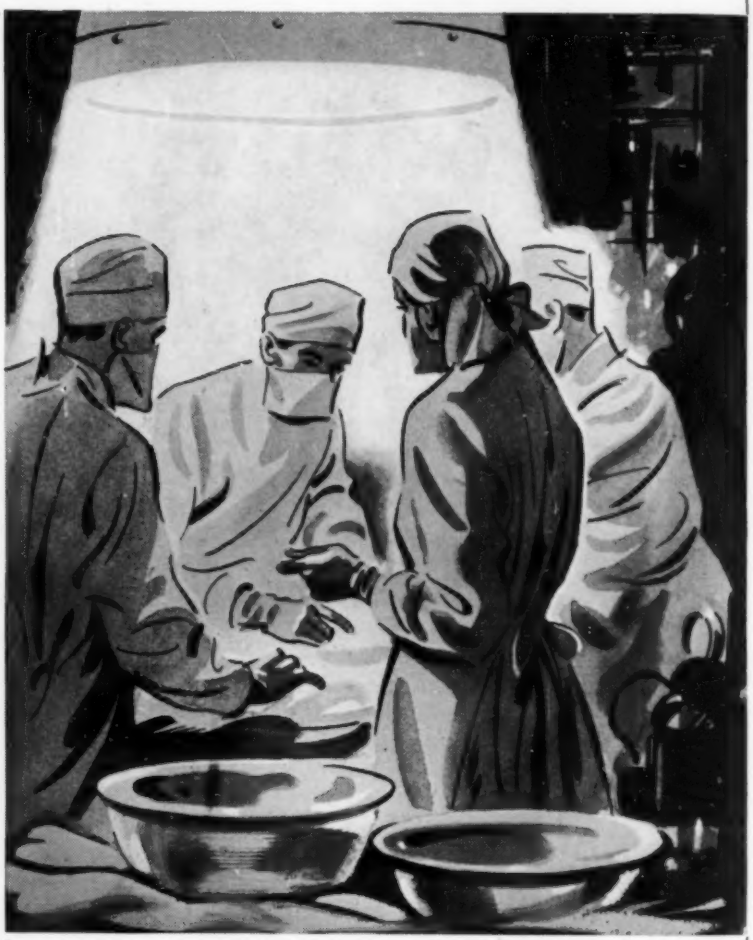

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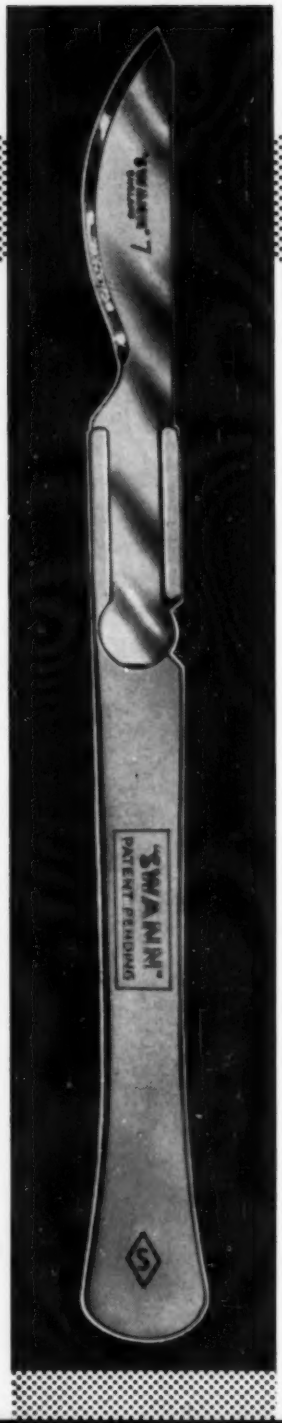

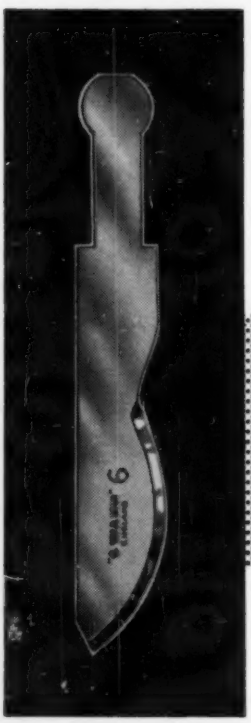

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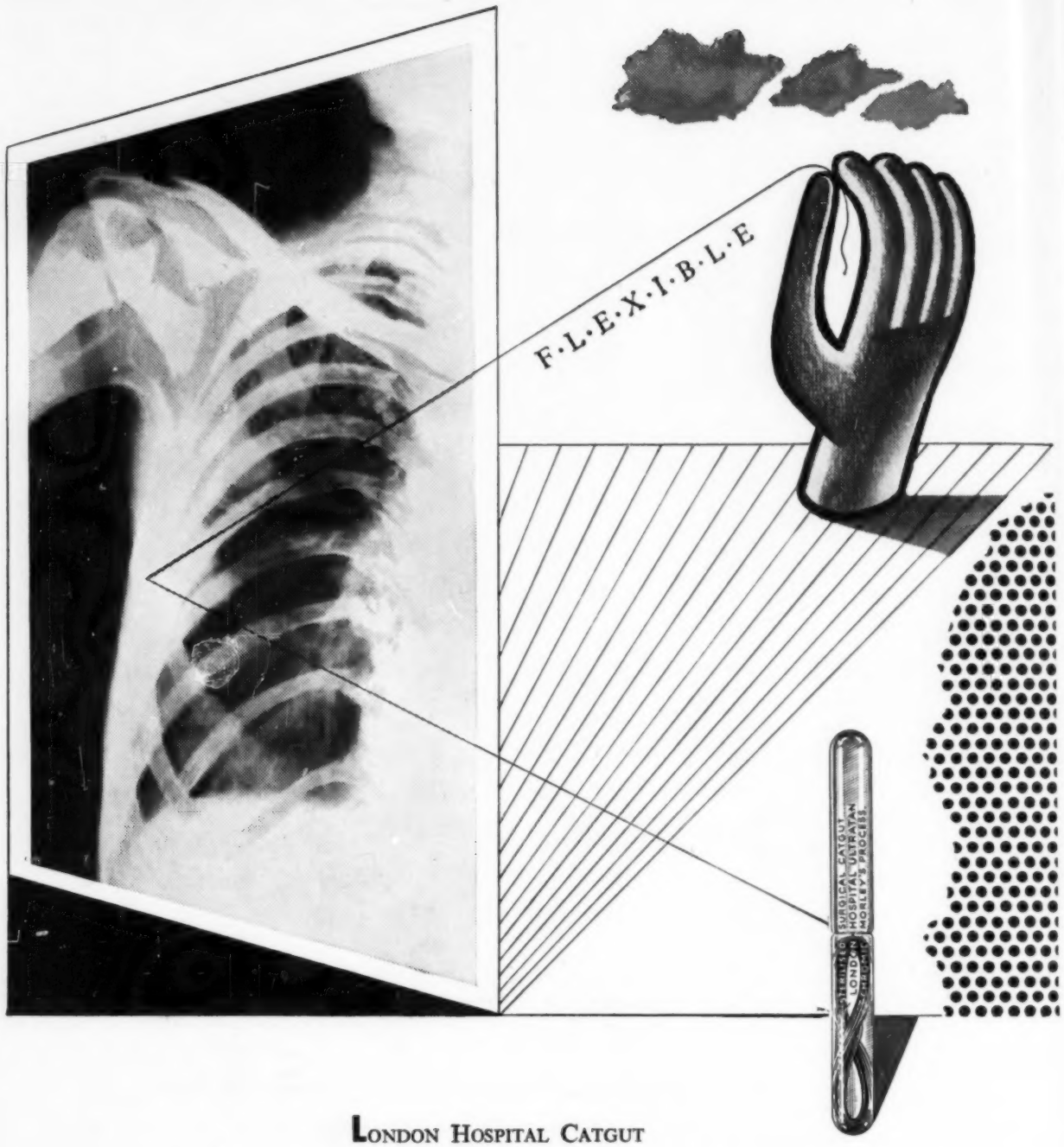

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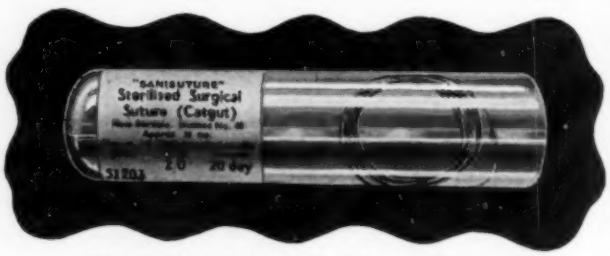

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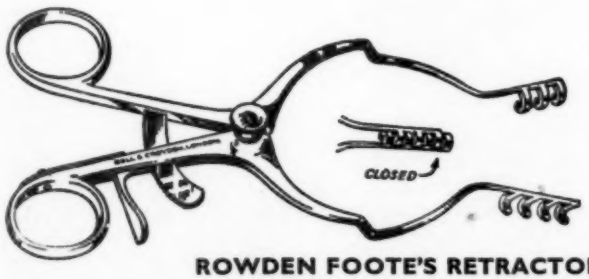

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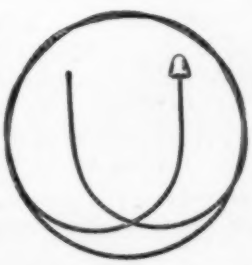

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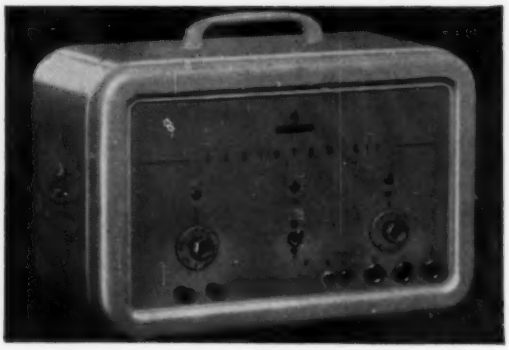

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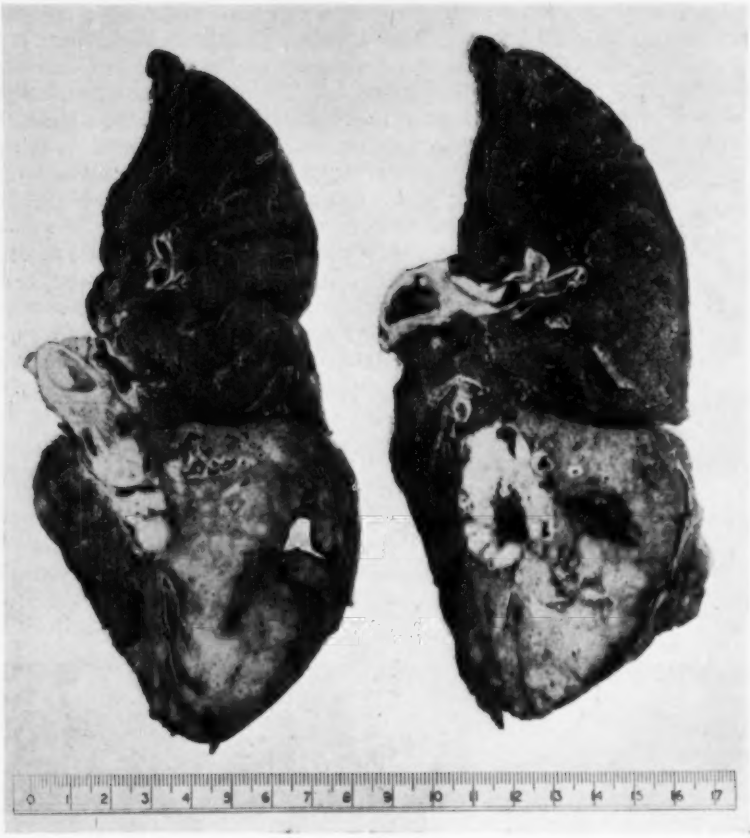

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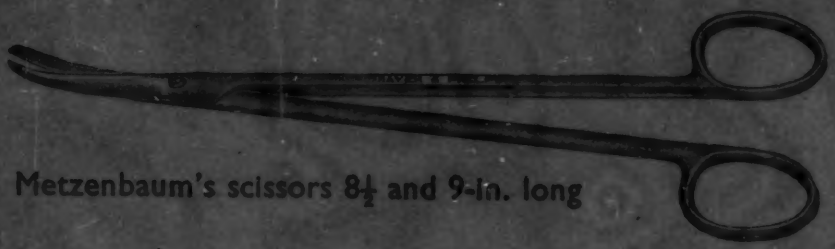

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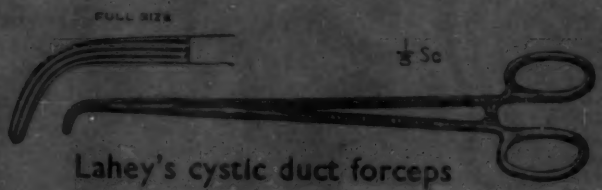

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